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VIII. HUTCHINSON'S TRIAD: THE TEETH

JONATHAN HUTCHINSON, born in 1828, died 1913, came of a Quaker family in Yorkshire. He settled in London after an apprenticeship in York, became attached to the Moorfields Eye Hospital in 1862, was Surgeon to the London Hospital until 1883, and was President of the Royal College of Surgeons of England in 1889. Observant and full of ideas, he soon realized that syphilis was being treated to relieve symptoms not to cure the disease. The actual cause was unknown, but as early as 1876 he stated that it was due to a specific and living microbe, contagious and transmissible only so long as it retained its vitality. " Some one will see it one day for it is beyond doubt that it must be there." That day he lived to see, for on March 3, 1905, Fritz Schaudinn showed Erich Hoffmann that spirillum which was called afterwards *Spirochæte pallidum.*

Hutchinson was insistent that syphilis needed prolonged treatment and was a pioneer in the administration of small doses of grey powder for many months, but never to salivation. His treatment, therefore, was in direct opposition to the method of the majority of his contemporaries, who gave large doses of corrosive sublimate only so long as the symptoms or signs of the disease were present.

As Surgeon to the Eye Hospital he was early interested in interstitial keratitis, which was not then uncommon, and he observed that many of these patients also had malformed teeth.

The first-fruits of his observations appeared in the paper contributed to the Pathological Society, May 10, 1858 (*Trans. Pathol. Soc.*, 1857–8, ix, pp. 449–456). It was referred to a Committee to report, the Committee consisting of Dr. Grailly Hewitt, Mr. Henry Lee, and (Sir) Henry Thompson, who sent in a guarded report dated July 14, 1858. Hutchinson says :—

" For a considerable time past I have been in the habit of recognizing in a certain very peculiar development of the permanent teeth an indication that their possessor had in infancy suffered from hereditary syphilis. A remark to this effect, which I made at a meeting of the Pathological Society early in the past session being received with expressions of incredulity, it occurred to me, that it might be well to make public such evidence as I possessed on the subject. With that view, the facts which are the basis of the following report were

collected. My friend, Mr. Coleman, our dentist at the Metropolitan Free Hospital entered with zeal into the subject, and readily agreed to take casts of the teeth of any patients I should send him.

" Most of the cases taken, were those of patients attending at the Royal Ophthalmic Hospital on account of chronic interstitial keratitis, an affection which is, I believe, almost always a result of inherited syphilis. Their ages varied from twenty-eight years to five years. In all a clear history of syphilis was established, either by the free confession of the patient's parents, or by the account given of

FIG. 319.—Hutchinson's teeth. (*From Power and Murphy's 'A System of Syphilis', and by kind permission of Dr. G. F. Still.*)

symptoms of undoubted character during infancy. The number of casts taken was thirteen and in all instances a stereographic portrait of the patient was also secured, which in many instances, illustrates very well the peculiar physiognomy impressed by the disease. With these remarks as to the nature of the evidence I will now pass to the conclusions arrived at.

" *That there is a peculiar condition of the teeth, which results from the influence of hereditary syphilis and that the most frequent features of this condition are the following :—*

"*a. Smallness.*—The teeth stand apart with interspaces, and are rounded and peggy in form, instead of flat.

"*b. Notching.*—They usually exhibit in their border a broad shallow notch, or at times, two or three (serrated). Owing to their softness; these teeth rapidly wear away, and this notching is thus often obliterated, but when markedly present, it is one of the most decisive conditions.

"*d. Colour.*—Instead of the clear smooth, white exterior of good teeth, they present a dirty greyish surface, totally destitute of polish and rarely smooth. No amount of cleaning will materially alter this feature which owes its existence, I believe, to the great deficiency of enamel.

"*e. Wearing down.*—As before observed their softness from deficiency of enamel renders them liable to premature wearing down. The teeth of a syphilitic patient not twenty will often be ground down as much as those of a very old person should be, and this in cases in which there is no peculiarity as to position, such as the front teeth meeting in the bite. An unusual degree of wearing down if coexistent with other peculiarities is then a suspicious condition.

"*f.* The signs mentioned apply almost exclusively to the incisors and canines, and in fact the grinders are usually altered in a very much less degree. Their surfaces are often more uneven than those of healthy teeth, and now and then they present tubercular projections of very peculiar character.

"These conditions are totally distinct from those produced by the ravages of caries. Very often the mouth of an hereditarily-syphilitic patient will present a full set of teeth quite free from decay, but all of them marked with features of unmistakable peculiarity. As a general rule, however, they are very liable to caries. Of course it is only when several of the conditions enumerated exist together, that a confident opinion can be given, and then it is the regularity of the type, and the fact that it marks all the front teeth in both jaws, which offers the best means of distinguishing it from other forms of disease or irregularity.

"The above remarks apply only to the permanent set. The milk-teeth of syphilitic-infants, are liable to exfoliate before being cut, and after having been cut, are often of small size, bad colour, and very liable to decay, but the notching and the peg-like form are rarely noticed."

SPONDYLITIS ANKYLOPOIETICA

(*Spondylitis Ossificans Ligamentosa*)

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THE condition considered in this report is that affection of the spine associated with the names of von Bechterew, Strümpell, and Pierre Marie. It has been shown by Buckley[5] and Llewellyn[24] that this condition was described most ably by an English physician, Dr. Bernard Connor, in 1700, more than one hundred years before the work of the continental authors to whom credit is given.

At the present time the word ' spondylitis ' (qualified by the type of organism which is considered responsible) is used as a generic term for lesions of the spine. In the case of ankylosing spondylitis, it has become the custom in some quarters to differentiate types of the disease according to the situation to which symptoms are referred at the outset of the disease, associating with these types the names of one of the above authorities. Thus in the Bechterew type the upper dorsal spine, and in the Marie-Strümpell type the lumbar spine is the first site where rigidity appears, and in spondylitis rhizomélique (Marie and Leri[17]) the large proximal joints are also involved. In this investigation it appeared that the Marie-Strümpell and rhizomélique spondylitis were the same disease, the sub-division being justified no doubt for descriptive purposes.

It has been difficult to interpret what is understood by the Bechterew type from the literature on the subject. Bechterew[4] described a fixed dorsal kyphosis with meningeal involvement and degenerative changes in the cord, associated with symptoms of paresis and altered sensation in the appropriate segmental region. Marie and Astie[16] showed in addition the influence of trauma and heredity in these patients. Few of the patients who are described in the recent literature conform with this description, and in this investigation a clear type which could be classed as ' Bechterew's disease ' was not recognized.

It is not denied that spondylitis ankylopoietica (Zigler[23]) affects the dorsal and cervical regions ; in fact the early clinical evidence is often a dorsal kyphosis, which is more or less fixed, but from the radiological examination there is rarely evidence of the cause of this kyphosis, or alternatively when calcification of ligaments is present in this region, it will usually be found to be present in a more marked degree in the lower thoracic and lumbar spine.

ETIOLOGY

In common with many of the conditions classed as rheumatic disease, the etiological factors of spondylitis are uncertain. It is customary to refer to toxæmia (Marie and Leri[17]) and the influence of temperature and trauma (Kaufmann[14]). Other observers have considered the disease an example of a metabolic disorder,

and recently the parathyroid glands have been incriminated and have been removed in some patients (Hoffmeister,[11] Oppel,[18] and Ballin and Morse[2]). Others have stated that the disease is a form of rheumatoid arthritis which attacks the spine (Jones,[13] Knaggs[15]). The theory which has found most favour is that the disease is of infective origin, but direct evidence to support this in all cases is difficult to obtain.

No writer has produced evidence that spondylitis is more common in the manual than the sedentary worker : 93 per cent of patients in this series lived within fifteen miles of London and 73 per cent led sedentary lives ; 23 per cent were manual or outdoor workers such as policemen, gardeners, etc. Only 4 patients suggested in their history that they had been subjected to any particular strain or exposure to which the condition might have been attributed.

Radiographs were secured of 124 patients, 106 males and 18 females. Of these, 79 males and 12 females presented the clinical and radiographic features of spondylitis ankylopoietica. The remainder, 27 males and 6 females, had sacro-iliac joint disease without changes in the spine ; in spite of this, 6 bore a clinical diagnosis of spondylitis ankylopoietica.

Holbrook[12] and others have remarked on the high percentage of males affected by this disease. Of their 22 patients, all were males and all were protected types, e.g., dentists, accountants, etc. In 1912 Dardel[6] stated that only two instances had been described in females, one by Gasne and one by Aldaroff.

Of this series, the age was known in 114 individuals. These were divided into two groups : Group I, with a clinical or radiographic diagnosis of spondylitis ; and Group II, with changes in the sacro-iliac joints alone. The average age of Group I, when presenting for treatment, was 36 years, and the average age when symptoms began was 25 years. The average age of Group II at the corresponding times was 28 years and 23 years respectively ; there is a fairly close resemblance between the last two figures in each of these groups.

Six individuals were excluded from Group I. These were patients with advanced calcification of spinal ligaments ; in each instance the patient gave a history of less than six months' duration. It was obvious that this could not be true, as it was doubtful if the changes which were visible could have been produced in as many years, and on these grounds they were excluded.

The high incidence of this disease in the young adult male militates against a metabolic origin, since at this time metabolic disturbances are notoriously rare.

Based on the work of Oppel,[18] of Leningrad, parathyroidectomy has been performed by surgeons in Germany and America. Oppel says : " The results of this operation may be considered very encouraging, for it primarily destroys the hypercalcæmia, thereby stopping the process of progressive ankylosis, and this is indeed the most important principle to be affected by the operation." Quoted by Oppel is Belsgorodsky, who found that all his patients suffering from ankylosing polyarthritis had an increase in the calcium content of the blood. In Oppel's own patients, 28 out of 42 had a raised blood calcium value, while 14 had a normal level.

Hoffmeister[11] describes the result of parathyroidectomy on a patient with advanced spinal rigidity, the cervical spine alone being stiff but not fixed. The blood calcium was reduced by two operations from 18 mgrm. to 14 mgrm. to 10 mgrm. per cent, and the blood phosphorus from 3·4 to 3 mgrm. per cent.

Ballin and Morse[2] state that "Multiple ankylosing arthritis should also be included in the chapter on parathyroidism. The metastatic calcification around infected joints, especially vertebral joints, is based on a moderate hypercalæmia."

The blood of a number of these patients was examined by Dr. Shackle at the British Red Cross Clinic for Rheumatism. The figures for the blood calcium were :—

1.	12·9	mgrm.	per	cent.	No spinal changes. Sacro-iliac joint involved
2.	12·1	,,	,,	,,	No spinal changes. Sacro-iliac joint involved
3.	12·6	,,	,,	,,	No spinal changes. Sacro-iliac joint involved
4.	11·0	,,	,,	,,	Early calcification of ligaments. Sacro-iliac joint involved
5.	11·4	,,	,,	,,	Moderate calcification of ligaments. Sacro-iliac joint involved
6.	12·1	,,	,,	,,	Moderate calcification of ligaments. Sacro-iliac joint involved
7.	12·6	,,	,,	,,	Moderate calcification of ligaments. Sacro-iliac joint involved
8.	9·5	,,	,,	,,	Advanced calcification of ligaments. Sacro-iliac joint involved (No evidence of activity)

In so far as these figures are of any significance, they confirm a slight hypercalcæmia in some patients, especially in the earlier types. In a few instances the inorganic phosphorus was estimated in addition :—

1. 11·6 mgrm. per cent (Ca). 3·76 mgrm. per cent (P)
   Advanced calcification of ligaments, sacro-iliac joint involved
2. 11·8 mgrm. per cent (Ca). 3·35 mgrm. per cent (P)
   Advanced calcification of ligaments, sacro-iliac joint involved
3. 10·1 mgrm. per cent (Ca). 5·8 mgrm. per cent (P)
   No spinal changes, sacro-iliac joint involved

There have been many patients in this collection whose history and clinical examination revealed a focus to which the disease might have been reasonably attributed, either in the form of gonorrhœa, ulcerative colitis, or septic tooth or antrum. But there was a lack of uniformity in these findings. Many individuals appeared quite free of infective foci.

Of these conditions, gonorrhœa and its sequelæ have been given great prominence (Whitman,[22] Jones and Lovett[13]). In this series only 9 patients gave a definite history of gonorrhœa. It is not suggested that this in any way represents the true number who had been affected by this disease. The investigation was not carried out with this point in mind, largely because of the difficulty in proving or disproving a past infection many years after the subsidence of the disease. In this connection it may be mentioned that iritis of a type which tended to recur (one patient had had four attacks) was present in 5·6 per cent of instances. There appeared to be some relationship between this and a past gonorrhœal infection, but the number was too small to draw any useful conclusions. The gonococcal complement-fixation test was carried out in a number of instances. A positive result was a rare event and the case was not included unless corroborated by the history.

The blood sedimentation rate was examined in a number of individuals. These were divided into two groups: Group I, with sacro-iliac joint changes and ligamentous calcifications; Group II, with sacro-iliac joint changes alone. It can be seen that in Group I the values are more consistently raised and that Group II shows peculiar variations. Some of these patients were examined more than once over a period of two years, and in these circumstances the average value is given :—

Group I		Group II	
1. 58 mm.		1. 110 mm.	
2. 100 mm.		2. 123 mm.	
1. 9 mm.		1. 6·5 mm.	
2. 26 mm.		2. 14 mm.	
1. 63 mm.	Average of 2	1. 23 mm.	Average of 3
2. 92 mm.	examinations	2. 50 mm.	examinations
1. 38 mm.	Average of 2	1. 5 mm.	
2. 68 mm.	examinations	2. 13 mm.	
1. 40 mm.		1. 75 mm.	Average of 7
2. 75 mm.		2. 110 mm.	examinations
1. 78 mm.		1. 57 mm.	
2. 110 mm.		2. 90 mm.	
1. 25 mm.			
2. 52 mm.			
1. 22 mm.			
2. 41 mm.			
1. 26·4 mm.			
2. 49 mm.			
1. 31 mm.			
2. 60 mm.			

There is a certain similarity in the radiographic appearances of rheumatoid arthritis and spondylitis, especially when the smaller joints are involved, but it does not appear that this is sufficient at present to justify the assumption of their common origin.

The calcification of ligaments extending some distance from the joints, as seen in the spine, the rib articulations, and hip-joints, is not a feature of rheumatoid arthritis. The age and sex incidence of the two are also dissimilar and there is no explanation of the fact that the spine is involved so seldom in rheumatoid arthritis and the minor joints so infrequently in spondylitis.

PATHOLOGY

Marie and Leri[17] considered that spondylitis rhizomélique began as a rarefaction of vertebral bodies which then produced a reactive hyperossification of ligaments. Simmonds[20] believed the most important feature was the ossification of ligaments surrounding joints and extending some distance from these joints. Ehrhardt[7] postulated a syndesmogenous synostosis of all vertebral joints with ossification of ligaments.

The German workers, amongst whom are Siven,[21] Fraenkel,[9] Rumpel,[19] Beadle,[3] and Schmorl, believe the process begins in the capsule and articular surfaces of the intervertebral facets in the form of an ulcerating disease. Several patients have been examined at autopsy. Siven[21] in 1903 reported four instances, of which one was examined at autopsy. He found the most advanced changes in the joints between the ribs and the spine, where there was total ankylosis, and the intervertebral discs were only slightly diseased. He concluded that it was an inflammatory disease of the joints which led to complete ossification of the spine.

Güntz[10] in 1933 summarized the present view of this school. From the post-mortem examination of an early case and correlation with nine patients with advanced disease in Schmorl's collection of spines, he concluded that the disease consists of an inflammation of the small joints of the spine, with round-cell infiltration, hyperæmia, and, above all, connective-tissue formation. The final result is ossification of articular surfaces. All stages of the disease are found in different joints.

Some joints may escape. No changes are found in the bodies of the vertebræ in early stages, and the changes which take place in these structures and in the ligaments during the course of the disease are of a secondary nature. Unfortunately the radiographs of this patient were not published; it would have been an advantage to see the stage of the disease as described.

The information gained from a post-mortem examination is greater than can be obtained from any number of radiographs, but there is no record of the post-mortem condition of the sacro-iliac joints in these patients. This important point has been overlooked. Yet radiographs reveal that in every instance these joints are involved.

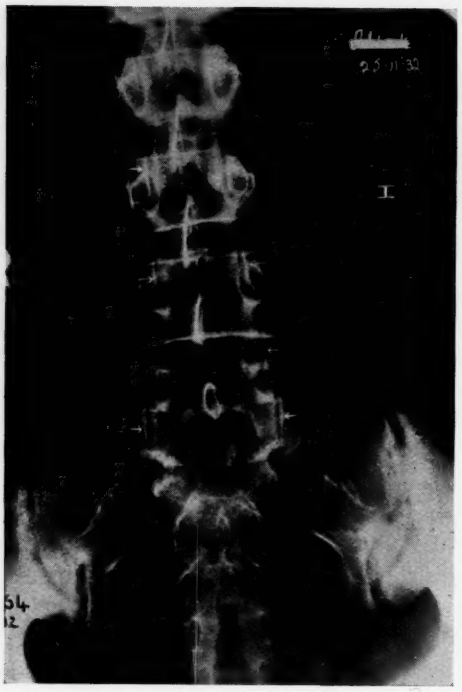

FIG. 320.—An example of sacro-iliac joint disease. There is irregular destruction of the joint margins, with some sclerosis of subarticular bone. Note the intervertebral joints, most of which show clearly and do not appear to be involved. There is an absence of calcification of ligaments.

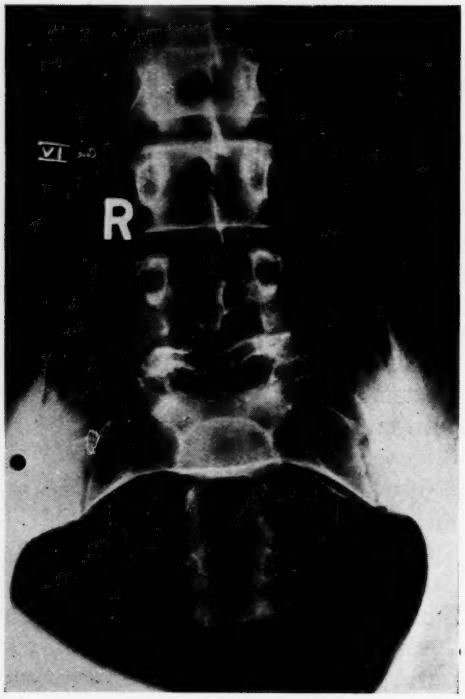

FIG. 321.—A rare type with very marked sclerosis of bone associated with a long history. No calcification of ligaments.

From the study of the radiographs I am of the opinion that the changes in the sacro-iliac joints precede destruction of the cartilage of the intervertebral facets and precede calcification of ligaments. It can be contended that the facets are involved before or at the same time as the sacro-iliac joints, but owing to the limitations of radiographic technique this involvement cannot be shown. It is true that the radiograph will only show gross pathological changes in the facets, but there were many cases in the series in which there was well-marked sacro-iliac disease with apparently normal intervertebral joints, and others with a longer history in which both the sacro-iliac and intervertebral joints are obviously involved. As described by Güntz, normal and diseased intervertebral joints were seen in the presence of

calcification of ligaments, especially in the early stages of calcification. In this series it was noted that these normal facets were the more rare, the more advanced the calcification of intervertebral ligaments. In the last stage, i.e., the bamboo spine, no normal facets were found. (*Figs.* 320–337.)

THE SACRO-ILIAC JOINT

It has been shown that the sacro-iliac joint is not a synchrondrosis since it possesses the essentials of a true joint (Albee[1]). It is affected as a secondary process in many diseases, of which tuberculosis is probably the most common. It is also subject to many congenital and age variations, and the lower edge of the ilium is frequently grooved by large blood-vessels entering the pelvis, producing an appearance of lipping of this edge, simulating osteo-arthritis. The radiographic interpretation of these joints is extremely difficult and all the aids of radiographic technique are necessary to produce good results.

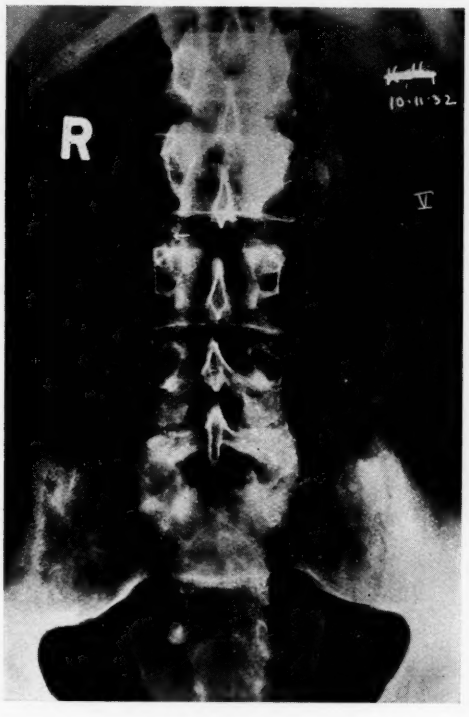

FIG. 322.—Nov. 10, 1932.

FIG. 323.—Feb. 26, 1935.

FIGS. 322, 323.—Showing progressive calcification of ligaments of the spine associated with sacro-iliac joint disease. Note that the only intervertebral facet which can be seen appears irregular. The others are probably obscured by ankylosis.

One or more authors in France, Germany, America, and England have remarked on the fact that the sacro-iliac joints tend to be involved in a certain percentage of instances of spondylitis ankylopoietica. When examining these radiographs it was

FIG. 324.—Feb. 19, 1930.

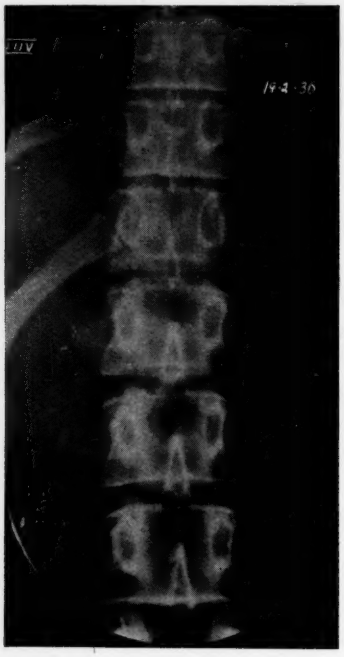

FIG. 325.—Feb. 19, 1930.

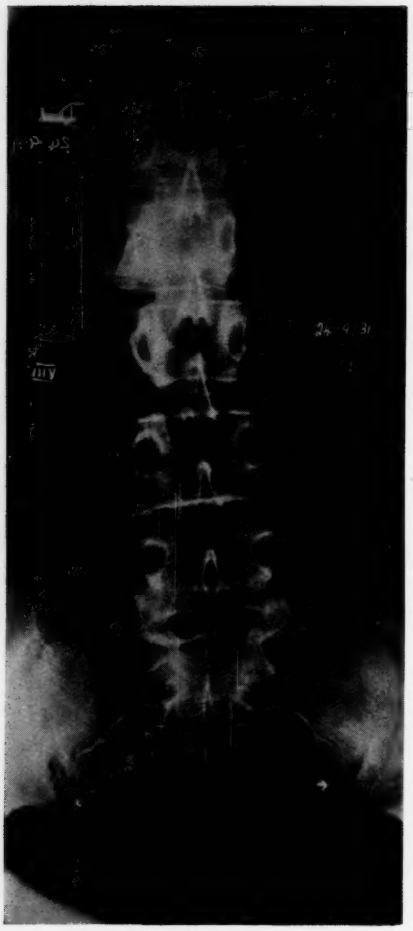

FIG. 326.—Sept. 24, 1931.

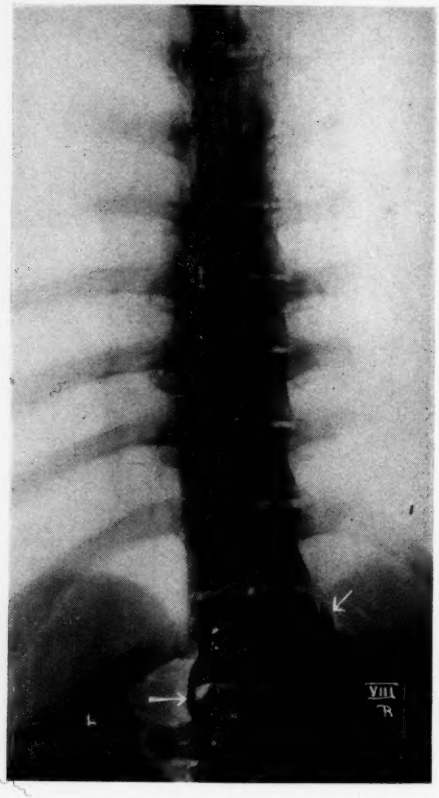

FIG. 327.—July 30, 1935.

found that the sacro-iliac joints were involved in all instances in which there was the typical calcification of ligaments of spondylitis, and it was noticed that this was the case in even the earliest examples—that is, those with only a few strands of calcification between vertebral bodies. A few individuals were found also with what appeared to be the same process in the sacro-iliac joints, but no calcification of ligaments. Several of these were found to have rigidity of the spine on clinical examination, and others complained of generalized pains without symptoms in the back. In every instance the radiographic appearances indicated the same disease process in the sacro-iliac joints, early in some, more advanced in others.

FIG. 328.—July 30, 1935.

FIGS. 324–328.—The sacro-iliac joint disease can be seen in the radiograph taken of the hip on Feb. 19, 1930. At this time the whole spine was radiographed and is clear. Unfortunately no further films were made of the sacro-iliac joints at this time. On Sept. 24, 1931, there is still no calcification of ligaments apart from a doubtful lesion between Thoracic XII and Lumbar I; the changes in both sacro-iliac joints can be seen. In the last film, early calcification of ligaments can be seen at the site which was previously clear. The sacro-iliac joints are now ankylosed.

The changes in these articulations consist of an irregular destructive process affecting the joint surfaces, and the clear definitition of the anterior and posterior edges of the joints, as seen on a radiograph, become blurred and serrated. Next the cartilage space is destroyed and slight sclerosis becomes evident in the peri-articular bone of the sacrum and ilium. The amount of destruction and sclerosis varies considerably in different individuals, but both proceed until the joint is destroyed, when ankylosis takes place; the sclerosis then becomes less marked and eventually there is continuity of bone between the sacrum and ilium with little or no trace of the joint.

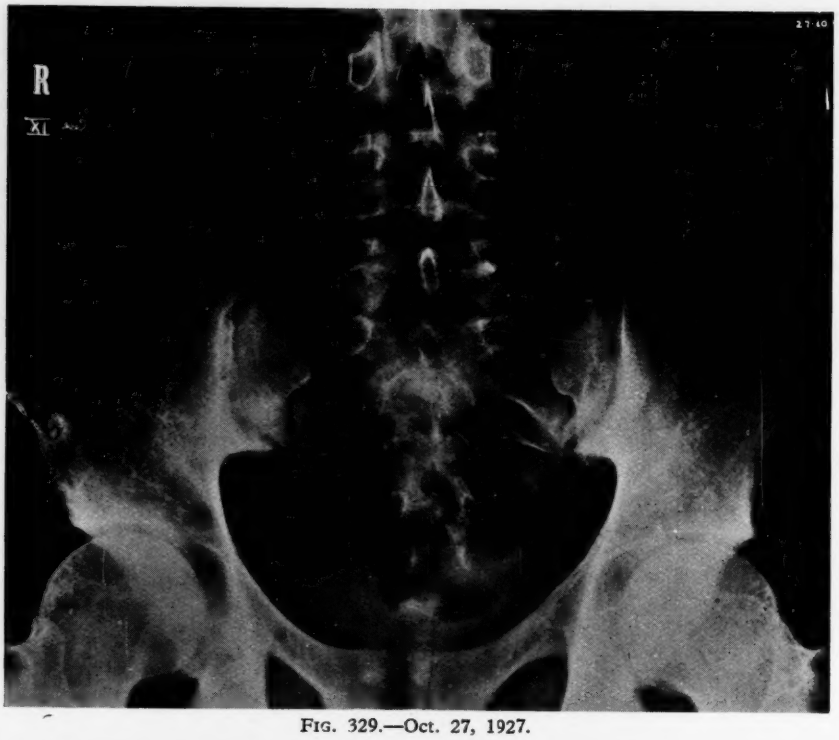

FIG. 329.—Oct. 27, 1927.

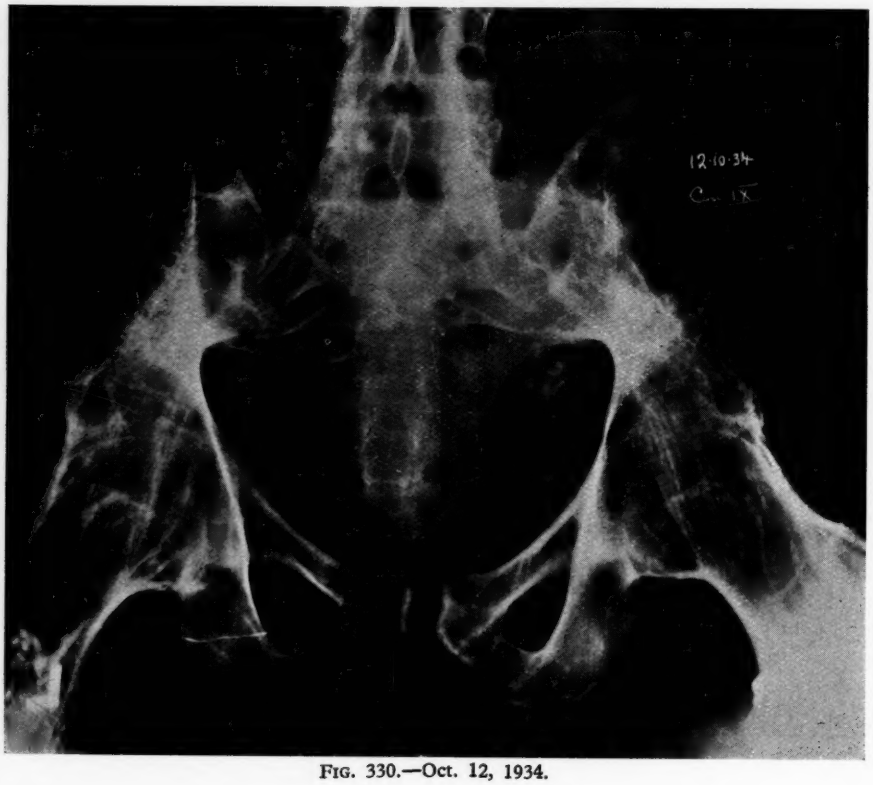

FIG. 330.—Oct. 12, 1934.

At present the stage of ankylosis represents the customary state of affairs when the changes of spondylitis ankylopoietica are manifest and the diagnosis of this disease is made.

It has been mentioned that the destructive changes which precede ankylosis, before there is calcification of ligaments, are also believed to be manifestations of

FIG. 331.—Oct. 12, 1934.

FIGS. 329–331.—In the first film (Oct. 27, 1927) both sacro-iliac joints are involved, showing a broad irregular joint space due to destruction of the joint margins. In the second film there is complete ankylosis of both these joints and the hip-joints. There is also advanced calcification of the lumbar ligaments. The last film (also Oct. 12, 1934) is included as it shows the so called 'tram-track' type of calcification of ligaments. Fortunately the patient had kept his earlier radiographs and it is possible therefore to show the earliest type of change in the sacro-iliac joints and the fully advanced disease. There is an interval of seven years between these examinations (*Figs.* 329, 330).

spondylitis or, more correctly, to be evidence of a prespondylitic condition. Most orthopædic surgeons and practitioners in physical medicine are familiar with patients in whom spinal rigidity is taken to indicate the presence of spondylitis ankylopoietica but in whom the radiographic examination reveals no visible changes in the spine.

Sir Robert Jones[13] has said in reference to the X-ray diagnosis of spondylitis: "In the absence of positive signs, the diagnosis must be made by exclusion and often a period of observation will be necessary before reaching a final conclusion." In other words, in its early development spondylitis passes through a period when the clinical history and physical signs are in favour of the diagnosis but this cannot be confirmed by radiology. It is suggested that radiographs should be taken of the sacro-iliac joints at this time.

THE CHANGES IN LIGAMENTS

This feature, consisting in the deposition of calcium in ligaments, thereby rendering them opaque to X rays, has been described by many writers. These changes may be seen in the anterior and posterior longitudinal ligaments and the

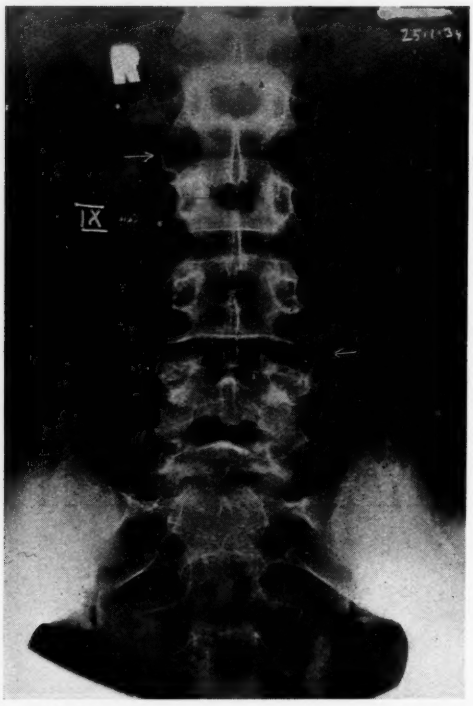

FIG. 332.—An example of the moderately advanced disease. The sacro-iliac joints are seen in the early stage of ankylosis with a trace of activity still present. Note that only the remains of the intervertebral joints can be seen.

ligamenta flava, producing a streaky appearance of the bones, especially in the lower lumbar region, often made more obvious by the associated decalcification of the bodies. Or there may be a 'tram-track' effect when the ligaments of the interarticular facets are calcified—namely, two opaque parallel lines on either side of the mid-line, or a single line in the median plane if the interspinous and supraspinous ligaments are affected. The 'bamboo spine' is produced by calcification of the ligaments round the intervertebral discs. These varieties are shown in *Figs.* 331 and 333. Other ligaments may show changes—for example, the radiate

ligaments of the heads of the ribs, the pubo-capsular and ilio-femoral ligaments in the hip, and ligaments of the pubic symphysis.

A survey was made of the radiographs to determine the most common site for the earliest changes in the spine. This was found to be between the vertebræ on either side of Thoracic XII and between Lumbar IV and Lumbar V. There were, of course, many exceptions to this statement, but this appeared to be the region of predilection, in spite of the prominent symptom of rigid dorsal kyphosis.

Other points noted were: the intervertebral discs were of normal width in spite of advanced changes in the ligaments around them; the decalcification of the bodies was often marked but was not necessarily an early sign; the streaky type of calcification was most obvious in the lumbar spine between Lumbar II and Lumbar V.

A peculiar manifestation of the disease was seen in some of the bones; it was difficult to decide whether this was a superficial osteitis without condensation or was due to changes in the ligaments at their insertion into bone. It was found, as a rare event, in advanced histories in the ischial tuberosity, the patella, and os calcis; seen apart from the spinal condition it would be most difficult to interpret correctly.

THE PRE-SPONDYLITIC HISTORY

A comparison was made of the early history of patients with fully developed spondylitis with those in whom sacro-iliac disease only was present. The march of symptoms which occurred early in the disease, described by those who had advanced changes in the spine when they came to examination, was strikingly similar to that in patients who had sacro-iliac disease only, and this suggested that there is a definite type of clinical history associated with this disease in the early stages. In this connection the age and sex of the patient is of considerable importance.

The pre-spondylitic history consisted of a number of attacks of pain of a fibrositic character recurring over several years, with free intervals. These pains were referred to the muscles or the neighbourhood of joints and were associated occasionally with a synovitis, which tended to resolve without deformity, especially in the peripheral joints. The site of muscular pains appeared to be most commonly in the thighs and buttocks, and varied from vague pain noticed especially on commencing movement after a period of rest, to severe attacks sufficient to incapacitate the individual. It was fairly characteristic that these pains, in any limb, tended to be in the proximal rather than the distal segments. The trunk was also affected, the complaint being made of 'tightness round the chest', etc. Many patients stated that these symptoms were worst when they were tired, or when in bed at night, interfering with sleep, and that they were relieved by exercise after a preliminary difficulty in commencing movements. A small number of patients gave a history of an acute onset with swelling of many joints, resembling rheumatic fever, but differing in that pain tended to persist after the swellings subsided. In such individuals ultimate limitation of movement was uncommon in these joints. The diagnosis of the true condition would appear to be impossible in this acute phase. Apart from this type, there were cases with a slow onset of swelling in one joint.

Distal joint involvement is believed to be a rare event in spondylitis ankylopoietica, and it was seldom present at the time of examination, but relying on the patient's statement, one or more joints had been swollen, presumably owing to

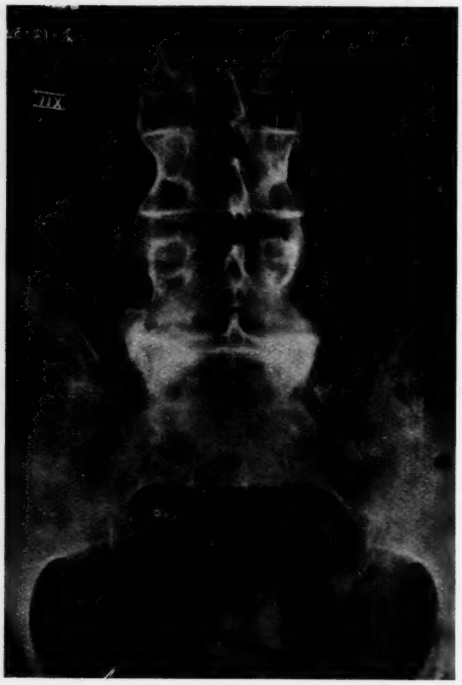

FIG. 333.

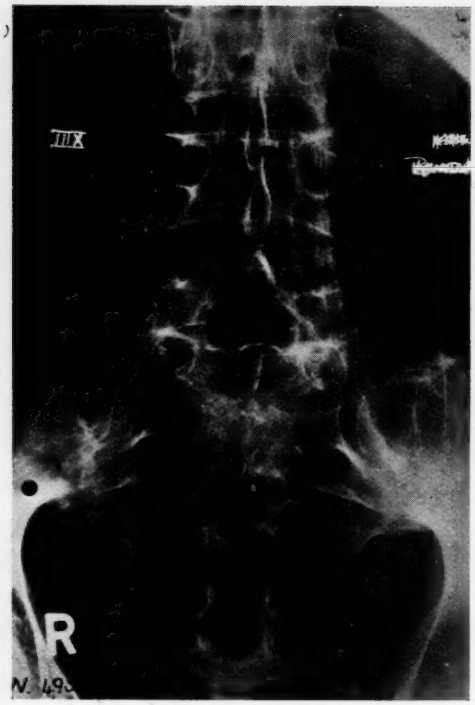

FIG. 334.

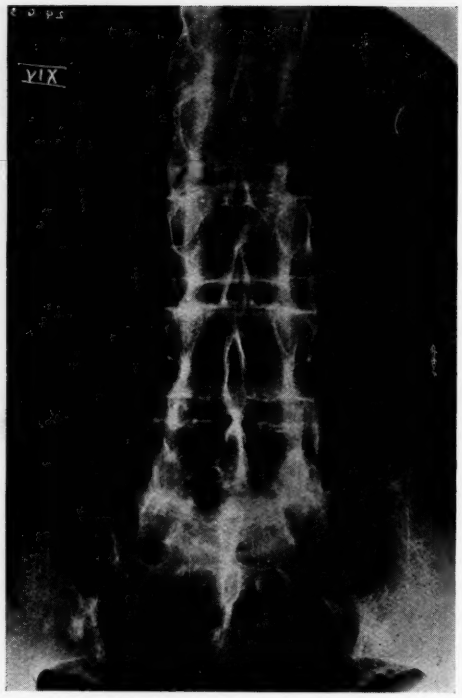

FIG. 335.

FIGS. 333–335.—The three types of end-result as seen in the lumbar spine: *Fig.* 333, The bamboo spine; *Fig.* 334, The 'streaky' type; *Fig.* 335, A combination of these two. The sacro-iliac joints are ankylosed. No intervertebral joints are visible.

synovitis, in 23 per cent of instances. The knee-joints were involved most frequently, the sternoclavicular joints in only two cases.

Restricted movement was rare in the distal, but common in the proximal, joints; it tended to be bilateral, and was present in 13·7 per cent in the shoulder and in 12·9 per cent in the hip; often both were affected in the same individual.

There were two exceptional instances in which the hip-joints were attacked early, with a severe onset, accompanied by great pain. These patients lay in bed afraid to move, with the joints slightly flexed; although there were early spinal changes these were overshadowed by the condition in the hips, and the diagnosis was not made until radiographs were taken.

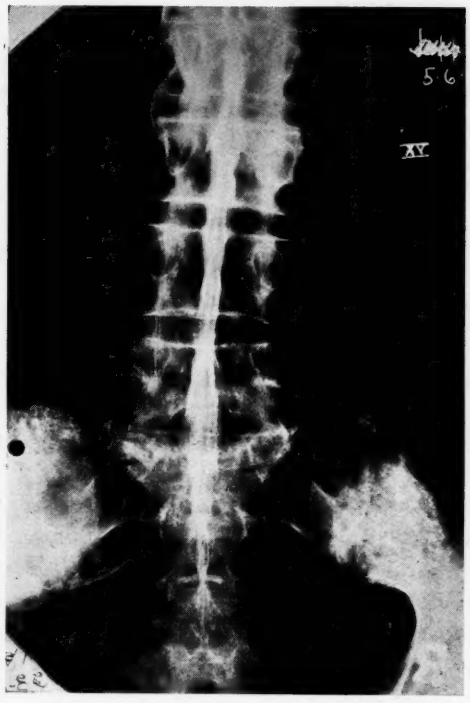

FIG. 336.

FIG. 337.

FIGS. 336, 337.—*Fig.* 336 is an example of the predominant calcification of the interspinous ligaments, simulating an Albee graft. *Fig.* 337 shows the changes, as described, which occasionally occur in the ischial tuberosity and elsewhere in bone. This is the same case as shown in *Fig.* 334.

Sciatica was fairly common as an early symptom, but it did not appear to differ from the ordinary interstitial neuritis. Of the whole series, 11·2 per cent had been diagnosed by doctors as sciatica at some time, 14·5 per cent as fibrositis or muscular rheumatism, and 7·2 per cent as infective arthritis. It is possible that these diagnoses were correct and that the early symptoms of spondylitis were due to inflammatory changes in fibrous and connective tissue.

After a varying period of pain in the limbs with or without remissions, the complaint began to be made of stiffness and pain in the back. This became more or less constant, and from this time onwards the diagnosis became more easy with each succeeding year.

In taking the histories of these people, one became so accustomed to the type of complaint which has been described under the ' pre-spondylitic period,' that it was difficult to believe the patient who stated that symptoms began in the back and not in the muscles and joints. Without any doubt this does occur, but it appeared to be more rare than the first type of onset.

So constant were these symptoms that it was believed to be a justifiable procedure to radiograph the sacro-iliac joints in any case of fibrositis in a young adult male who gave a history of recurring attacks which had not responded to treatment or had responded temporarily. After the age of 30 years the possibility of this examination proving of any value is more remote.

DIFFERENTIAL DIAGNOSIS

There are relatively few conditions which can be confused with spondylitis ankylopoietica. Spondylitis muscularis and senilis (Knaggs[15]) are variations of the same condition, due to muscular weakness with secondary dorsal kyphosis; there is some narrowing of the discs on the anterior border, calcification of ligaments is absent, and the vertebral bodies may be much decalcified. The late stage of kyphosis adolescentium does not present difficulty, since there is no calcification of ligaments, and the wedged irregular vertebral bodies in the later stages are typical of the condition.

The most important differentiation is from osteo-arthritis of the spine. There are several articles in the literature in which no distinction is drawn between these two conditions. In most instances of osteo-arthritis the patients are over fifty years of age or there is a history of strain, the so-called ' labourer's spine '. A paper has been written recently by Forestier and Robert[8] on the differentiation of osteophytes and calcification of ligaments.

If radiographs are taken, the diagnosis should not present great difficulty; usually most of the stages of these conditions can be seen on the single film, and the early formation of an osteophyte is unlike the calcification of ligaments. The osteophyte is frequently a thick, blunted, bony process projecting at an angle from the margin of the vertebra. The early calcification of a ligament is a thin-pointed homogeneous deposit running from one body to another in the form of an arc of a circle.

PROGNOSIS

The more or less hopeless prognosis of fully developed spondylitis is well known. The situation is more grave than in the average instance of rheumatism, since the sufferer is nearly always a young adult male who, after a varying period, is not able to earn a living. It appears that the condition does not progress invariably to the complete ' bamboo ' spine; some patients reach a stage of limited mobility, with subsidence of subjective symptoms.

It is difficult or impossible to form an opinion on the prognosis in early stages from the symptoms and radiographs of the spine. It has been noted all through this series that the most severe instances were those with the greatest destruction of the sacro-iliac joints, and that those with mild changes in these joints, often difficult to detect, had infrequent attacks of pain, insufficient to produce incapacity. It was observed also that the fully developed ' bamboo ' spine was always associated with

destruction of the sacro-iliac joints, with bony ankylosis so complete that the previous position of the joint could be determined with difficulty.

In contrast to this the condition in which there occurred calcification of a few ligaments only, which did not alter materially in two or three years, was usually associated with what was believed to be a mild process in the sacro-iliac joints, often accompanied by sclerosis of the subarticular bone.

The patients who answered the follow-up letters by saying they did not need to attend as they were cured or were progressing satisfactorily without treatment were all individuals who had what was interpreted as a mild or quiescent disease of the sacro-iliac joints. It is suggested as a reasonable conclusion that some idea of the prognosis can be gained from a scrutiny of the sacro-iliac joints.

TREATMENT

This subject is only considered from the general aspect of the course of the disease as shown by clinical and X-ray examination. It was evident that it will always be difficult to assess the value of any form of treatment in this disease.

Firstly, the normal advance of the condition is so slow that a period of years may be necessary to observe additional calcification of ligaments. It is necessary, therefore, to rely on clinical findings, such as the relief of pain, freedom of movement, gain in weight, etc. This is complicated by the fact that patients without treatment may have periods of relief and exacerbation. Some of these patients terminated treatment, as they felt they were cured or had obtained such relief that treatment did not appear to be necessary, only to return months or years later with a fresh attack. This applied more particularly to patients with only sacro-iliac joint disease.

Secondly, the disease sometimes appears to wear out, a stage being reached in which, although there is a fixed kyphosis, muscular pains occur infrequently and there is some recovery of general health. In this case the particular treatment which the patient is having at this time will receive credit which may not be justified.

CONCLUSIONS

The radiographs of 91 patients suffering from spondylitis ankylopoietica have been collected, together with the history and clinical findings. In many instances additional examinations have been made with further radiographs.

It is believed that the diagnosis of spondylitis ankylopoietica may be made earlier than at present.

From a detailed examination of the early history of these patients it was found that almost all gave a long history of muscular and joint pains before the onset of spinal rigidity. This type of history was so constant that it was thought to be possible to recognize it at the clinical examination.

It was also found that in spondylitis of this type pathological changes in the sacro-iliac joints were invariably present. There were no exceptions to this statement in 91 patients.

In addition, 33 individuals had pathological changes in the sacro-iliac joint without spinal changes. These patients gave a history of the same type as the above, and 6 had been diagnosed, in fact, as spondylitis ankylopoietica by the clinician before radiographic examination.

From the information which has been gained from this collection of patients it is believed that sacro-iliac joint disease of this type is the earliest manifestation of spondylitis and may be present as a useful diagnostic indication for several years before the back is involved.

In addition, the etiological factors have been considered, with the blood sedimentation rate and blood calcium in some of the patients.

It is thought that some indication of the severity and prognosis of the disease may be obtained by the degree of activity of the process in the sacro-iliac joints.

I wish to express my gratitude to Dr. Gilbert Scott, with whom the work was begun some years ago. The material was obtained largely from the British Red Cross Clinic for Rheumatism, London, and I am indebted to both the medical and lay staff for their generosity in allowing me to use the Clinic for the collection of these patients during the last year. I also wish to thank Dr. S. A. Smith for suggestions which he has offered in the preparation of this paper. This work was undertaken with a part-time and expenses grant from the Medical Research Council.

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AN IMPROVED TECHNIQUE FOR THE INTRODUCTION OF RADIUM NEEDLES IN THE TREATMENT OF CARCINOMA OF THE BREAST

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One of the greatest difficulties in the treatment of carcinoma of the breast by radium needles is to be sure that the needles when introduced lie parallel to each other. When the patient is prone—the position in which radium needles are usually introduced into the breast—the nipple is central or approximately central in position; but when the patient sits up, as she does in bed after the operation, if the breast is at all pendulous the nipple comes to lie at a much lower level. It follows from this that radium needles lying parallel to one another when introduced are no longer parallel when the patient is back in bed. The shorter the needles, the more likely is there to be this alteration in their relationship. The breast is radiated unequally; in one place there will be overlap, in another deficient radiation. In order to overcome this difficulty the following technique has been adopted :—

The various glandular regions, the axilla, supraclavicular fossa, infraclavicular region, intercostal spaces, and epigastric region are needled in the usual way. The technique adopted here is that usually described. It is performed at the same time as the breast is dealt with, but before the breast is needled. It is in the actual needling of the breast itself that the improvements have been introduced. Careful co-operation by the nursing staff is essential for the method to be described. The nurses and house men rapidly become interested and learn to carry out their part of the technique—a very important one—with the greatest skill.

On the day before the operation, when the patient is in bed, and before the breast has been prepared for operation, the sister of the ward or the house surgeon first carefully marks the outline of the breast on the skin with a skin pencil, including the tail of the breast extending up into the axilla. The area so marked out is carefully measured, and a piece of red paper is then cut out its exact size. This is pinned to the notes and bed-letter and is sent to the operating-theatre before the operation begins.

The breast is needled by means of long hollow trocars measuring $9\frac{1}{2}$ in. in length, with an outside diameter of $\frac{1}{10}$ in. and an inside diameter of $\frac{1}{12}$ in. Each has a lance point at the one end which is detachable, and is covered with a protective cap. At the other end is a flange and split pin with which the trocar is held and introduced, also detachable (*Fig.* 338). These hollow trocars will contain the radium needles. The ends are made detachable in order to facilitate the loading and unloading. They are introduced through metal channels situated 1 cm. apart on a square metal frame, the size of which is adjustable by means of bolts and screws at the four corners, and can be made to take the largest breast (*Fig.* 339).

Actually the instrument consists of two of these frames attached to one another 1 cm. apart, and with the grooves for the trocars running in two directions at right angles to each other. Thus when the trocars have been all introduced, there are two rows of them, each trocar lying 1 cm. apart from and parallel with its neighbour, and each row lying 1 cm. apart and at right angles to the other first row. A number of metal stops of assorted lengths, the largest being 3 cm. and

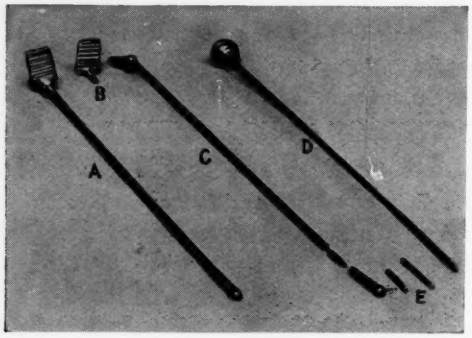

FIG. 338.—A, Trocar complete; B, Detachable flange; C, Trocar, showing detachable spear-point and cap; D, Stilette; E, Metal stops.

graduated in centimetres, and a stilette also graduated in centimetres and the length of the trocars, complete the apparatus (*see Fig.* 338).

The theatre sister receives the red paper outline of the breast, and placing it in the centre of the metal frame first adjusts the size of the metal frame until there is $\frac{1}{2}$ cm. between the edges of the breast outline and the sides of the frame. She then takes the hollow trocars and inserts them through the grooves in the frame

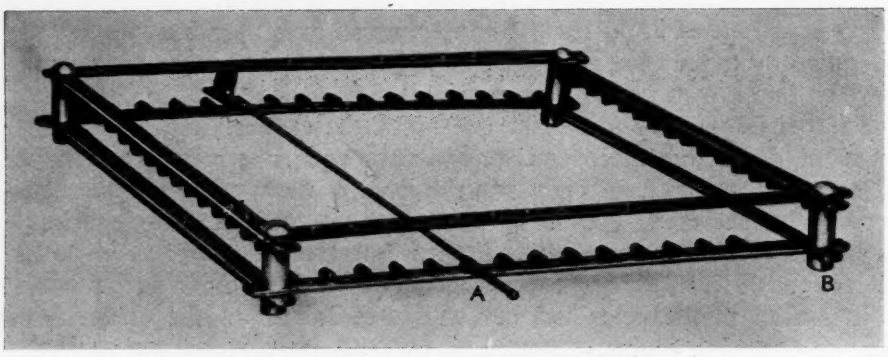

FIG. 339.—The frame. A, Trocar in position; B, Bolts for adjusting size of frame.

until the whole of the breast area has been covered. This manœuvre is repeated with the second row of trocars. The hollow trocars are now filled. Metal stops are introduced by means of the graduated stilette until the edge of the breast as indicated by the margin of the red paper is reached. Then radium needles are introduced throughout the length of the red disc, and when the other margin is reached, more stops until the trocar has been completely filled. The same process is repeated with the other trocars until in each row the portion of the containing

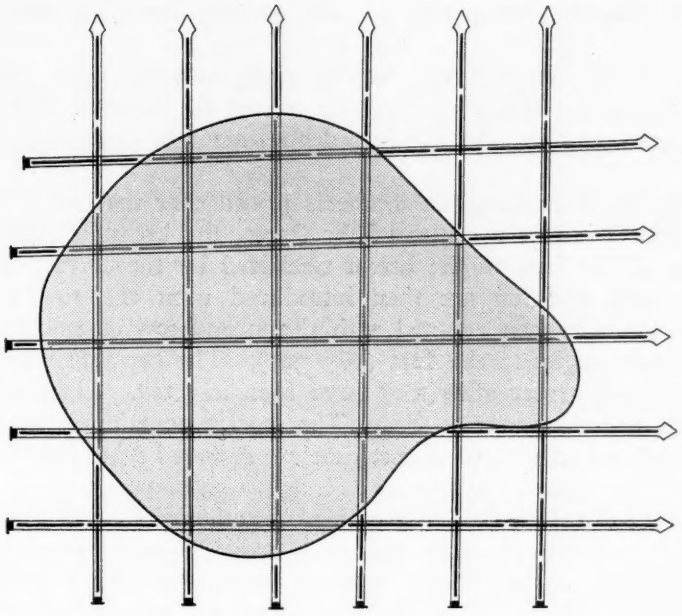

FIG. 340.—The parts of the trocars overlying the red paper are filled with radium needles, [the other parts with metal stops.

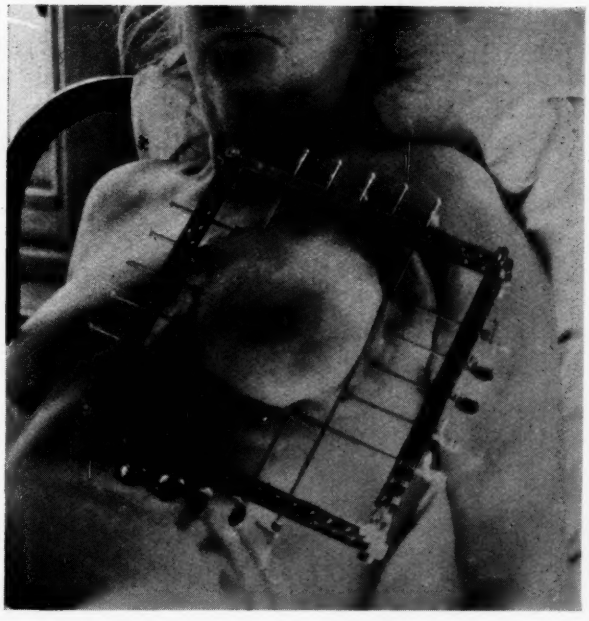

FIG. 341.—The frame applied to the breast, showing how parallelism of the needles is assured.

radium needles exactly corresponds to the portion overlying the red paper (*Fig.* 340).

The frame is then marked top, bottom, right, and left sides, and with the trocars *in situ* sterilized. When sterilized the trocars are removed from the frame, greased with sterile paraffin, and laid in order on a sterile cloth ready for use by the surgeon.

Under a general anæsthetic, the frame is placed over the breast, and, taking care that the nipple is in the centre of the frame, the breast is pulled forwards and the middle of the base of the breast transfixed by the correct trocar. The trocars to the right and left are then introduced until the row is complete. The same procedure is then adopted with the second row of trocars, which are introduced at right angles to the first (*Fig.* 341). The caps are placed over the pointed ends of each trocar after they have been inserted. Light dressings are applied and the patient returns to bed. The trocars containing the needles are left in for a week, and at the end of that time are removed quite painlessly without an anæsthetic.

The advantage of this method of needling over other methods is that parallelism of the needles is assured. Actually when the breast is pulled forwards, the area of the base diminishes so that the area radiated extends for a short distance beyond the breast margin. This is an advantage. There are no gaps in the radiation, and the whole of the base of the breast is uniformly radiated. Secondary radiation is not an important factor.

The whole operation takes only ten to fifteen minutes, as against thirty-five to forty minutes when each radium needle is introduced separately and each thread anchored to the skin by a stitch. The time-taking part of the operation—namely, the loading of the containers—is all done by the sister or house surgeon before the operation begins. The needles are safely boxed up in a container, and the risk of loss is reduced to a minimum.

When the demand for radium exceeds the supply, it is not always possible to obtain the long radium needles which are essential if a breast is to be needled in the usual way. With this apparatus needles of any length can be used. The apparatus is tolerated extremely well by the patient, and the discomfort is negligible.

FURTHER OBSERVATIONS ON THE DISTURBANCE OF METABOLISM CAUSED BY INJURY, WITH PARTICULAR REFERENCE TO THE DIETARY REQUIREMENTS OF FRACTURE CASES

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THE present writer has drawn attention to the marked loss of body nitrogen, sulphur, and phosphorus which occurs in the urine of otherwise healthy individuals who are in receipt of moderate or serious traumatic injury (Cuthbertson, 1930, 1931, 1932, 1934). This loss was observed to begin within a day or two following the injury, to reach a maximum within ten days, and then slowly to decline. It was noted that there generally occurred parallel increments in the basal consumption of oxygen, body temperature, and pulse-rate. It was suggested that this loss was probably more general than local, as in one fracture case 137 grm. N, equivalent to about 4·38 kilo. of muscle, were lost in the course of ten days. These phenomena were noted to occur in varying degree in uncomplicated fractures of the long bones, dislocations, effusions into joints, lacerations of soft tissues, and surgical incisions into knee-joints. In general the more severe the injury, the greater the catabolism. Other experiments indicated that the wasting of muscle and bone caused by immobilization, which of necessity occurs during treatment of fracture and knee-joint cases, was insufficient to account for the whole of this loss of substance, though it was undoubtedly a contributing factor. Although there was in most cases an evident initial depression of metabolism following the injury, the counter swing which followed was in excess of an immediate restitution.

As the accompanying fever was generally slight or scarcely appreciable in the milder cases, and as a study of pneumonia cases both before and after the crisis had failed to reveal catabolic effects in excess of those noted in severe injuries to the limbs, in particular those due to direct violence, it was considered that the loss of body substance was not proportional to the fever.

It was believed that these changes were the result of the organism catabolizing its reserves to meet the exigencies of repair and maintenance rather than due to the sweeping out of the disintegration products of the damaged tissues. The question naturally arose whether increased intake of foodstuffs could stem this loss of body substance or at least lessen its disintegration. The present communication is an attempt to determine this point: in addition certain other phenomena associated with injury are described.

EXPERIMENTAL

The patients were supplied with the requisite foodstuffs prepared in an appetizing form. It was generally found that after about a fortnight of high protein diet their appetite failed, and though the patients were still willing to co-operate they could not ingest the required amount.

Table I.—SUMMARY OF RESULTS OF EXPERIMENTS IN EIGHTEEN CASES OF FRACTURE

Case	Age	Period of Observation Reckoned from Day of Accident	Nature of Injury	Total Calories	N Intake (Grm.)			Maximum Urinary Excretion of N (Grm.)	Day of Maximum Urinary N Excretion Reckoned from the Time of Accident	N Excretion (Grm.) Excluding First Three Days of Observation			N Balance Excluding First Three Days of Observation (No. of Days)
					Basal N	Additional N and Source	Total N for Period (Average Daily)			Urine	Fæces (Average Daily)	Total N for Period	
1	43	2–14	Fractured humerus	1981	128·40	—	128·40 (14·27)	21·87	10th	154·90	11·52 (1·28)	166·42	— 38·02 (9)
2	59	3–14	Fractured humerus	1944	127·40	—	127·40 (14·16)	15·89	9th	135·01	8·64 (0·96)	143·65	— 16·25 (9)
3	24	2–17	Fractured tibia	1802	172·21	45·23 (Bovril)	217·44 (16·84)	36·18	7th	375·34	21·58 (1·66)	396·92	— 179·48 (13)
4	35	2–13	Fractured tibia and fibula and laceration of foot of one leg	914	34·92	85·05 (Lemco)	119·97 (13·33)	21·98	7th	171·92	8·28 (0·92)	180·20	— 60·23 (9)
5	23	1–15	Fractured tibia and fibula of one leg	1183	136·60	1·404 (Cystine)	138·00 (11·50)	29·10	9th	286·42	3·36 (0·28)	289·78	— 151·78 (12)
6	17	2–14	Lacerated tendo Achillis	2200	153·80	14·91 (Glycine)	168·71 (16·87)	19·31	6th	171·61	11·80 (1·18)	183·41	— 14·70 (10)
7	37	1–12	Fractured tibia and fibula of one leg	1669	109·80	25·20 (Glycine)	135·00 (15·00)	28·85	6th	219·78	6·45 (0·72)	226·23	— 91·23 (9)
8	21	2–22	Almost complete fracture of both bones of one leg	1990	196·20	47·88 (Eatan)	244·08 (13·56)	21·56	11th	338·65	17·46 (0·97)	356·11	— 112·03 (18)
9	20	2–14	Fractured tibia	3259	119·50	199·74 (Gelatin)	318·24 (31·82)	42·36	5th	370·36	12·48 (1·25)	382·84	— 64·60 (10)
10	25	2–16	Fractured tibia and	3097	117·84	347·40 (Gelatin)	465·24	43·31	7th	464·10	13·08 (1·09)	477·18	— 11·94 (12)

						(Gelatin)	(31·82)				(1·25)		(10)
10	25	2–16	Fractured tibia and fibula of one leg	3097	117·84	347·40 (Gelatin)	465·24 (38·77)	43·31	7th	464·10	13·08 (1·09)	477·18	− 11·94 (12)
11	23	2–27	Fractured humerus with bruising and laceration over supra-orbital ridge	2386	217·92	531·36 (Sodium caseinate)	749·28 (32·57)	38·51	6th	726·53	36·50 (1·59)	763·03	− 13·75 (23)
		5–14		2386	99·88	243·54 (Sodium caseinate)	343·42			365·38	17·63	383·01	− 39·59 (10)
		15–27		2386	118·04	287·82 (Sodium caseinate)	405·86			361·15	18·87	380·02	+ 25·84 (13)
12	26	2–12	Compound fracture of tibia and fibula of one leg	5392	79·55	227·70 (Sodium) caseinate)	307·25 (34·14)	42·39	11th	342·29	15·84 (1·76)	358·13	− 50·88 (9)
13	42	2–11	Fractured tibia and fibula at lower end of one leg	3557	166·74	—	166·74 (23·82)	30·69	6th	197·62	5·91 (0·84)	203·53	− 36·79 (7)
14	35	2–17	Compound fracture of tibia and fibula of one leg	3193	345·41	—	345·41 (26·57)	31·59	7th	354·99	17·68 (1·36)	372·67	− 27·26 (13)
15	38	3–15	Fractured tibia and fibula of both legs	3214	318·40	—	318·40 (31·84)	40·25	8th	318·89	17·10 (1·71)	335·99	− 17·59 (10)
16	23	1–18	Fractured tibia and fibula of one leg	3044	507·60	—	507·60 (33·84)	42·00	6th	523·72	29·70	553·42	− 45·82 (15)
17	44	2–21	Fractured tibia and fibula of one leg	3606	623·73	—	623·73 (36·69)	40·82	8th	609·78	39·44	649·22	− 25·49 (17)
18	31	2–13	Fractured fibula	4135	147·51	—	147·51 (16·39)	22·96	6th	172·05	11·36	183·41	− 35·90 (9)
19	16	1–8	Fractured tibia and fibula of one leg	3592	41·55	—	41·55 (8·31)	20·67	4th	80·12	9·00	89·12	− 47·57 (5)

Difficulty was experienced in deciding the period of time over which the net gain or loss of body substance should be assessed. It was eventually decided to omit the first three days of diet from the final computation. The experiments are arranged in groups, and, although not strictly chronological, this order is the more convenient. The analytical methods used were in general similar to those adopted in previous experiments.

Urinary K was determined by the method of Kramer and Tisdall (1921) with slight modification. After separation of the potassium cobalti-nitrite it was oxidized with a known excess of standard permanganate as in the original method, but the excess after oxidation was determined by the addition of KI (Kahlbaum) and titration of the liberated I_2 with N200 sodium thiosulphate.

Sodium in the urine was determined by the method of Kramer and Gittleman (1924) following the incineration of 50 ml. urine acidified with H_2SO_4 at as low a temperature as possible. The residue was dissolved in water and made up to 50 c.c.: 1 ml. was then taken for analysis.

Preliminary experiments (*Cases* 1–12) were designed to determine if procedures such as daily massage (*Cases* 1, 2), the ingestion of meat extractives (*Cases* 3, 4), additional amounts of cystine or glycine (*Cases* 5–7), hydrolysate of mixed ox tissue (*Case* 8), or the addition of large quantities of proteins such as gelatin (*Cases* 9, 10) or sodium caseinate (*Cases* 11, 12) could stem the loss of body N when superimposed on diets of low first-class protein content and generally low caloric value. It will be obvious from the later experiments (*Cases* 13–19) that no success could have resulted from these experiments, since the addition of large quantities of first-class protein to diets of high caloric content failed to maintain N equilibrium at the height of the catabolism.

Cases 1–8 were given diets of low caloric value in order to determine if the special agent which was being applied had an appreciable effect.

The Effect of Gentle Massage and Movements in the Treatment of Two Ambulant Cases with Fractured Humeri (*Cases* 1, 2).—*Cases* 1 and 2 were placed on diets of average first-class protein content (11·68 grm. and 14·16 grm. N per day respectively). In the course of nine days *Case* 1 lost 38·0 grm. N and *Case* 2 lost 16·2 grm. N. A kick from a horse fractured the humerus of *Case* 1 at the junction of the lower and middle thirds. *Case* 2 was injured at practically the same site by a fall of stone. There was also some bruising and laceration at the site of injury in the latter case. The daily fluctuations in the urinary volume bore a fairly close relationship to the total N excretion.

In these two cases disuse atrophy was reduced to a minimum by allowing the patients freedom to walk about hospital and by massaging and manipulating the injured area daily from the date of injury. It is evident that this procedure did not eliminate the loss of body N. These two cases were the oldest of the series, being 43 and 59 years respectively. This disparity may have been a factor in deciding the degree of catabolism.

The Effect of Meat Extractives (*Cases* 3, 4).—*Case* 3, who was on a moderately low first-class protein diet, received in addition 60 grm. meat extractive (Bovril) daily, equivalent to an extra 3·48 grm. N. *Case* 4, who was on a very low first-class protein diet, received daily 100 grm. meat extractive (Lemco), equivalent to 9·45 grm. N.

In the course of thirteen days *Case* 3 (fractured tibia at junction of middle

and lower thirds) lost 179·4 grm. N (equivalent to about a stone of muscle if that be the source of this N), and in nine days *Case* 4 (fractured tibia at mid point with extensive laceration of one leg, skin having to be removed off part of distal half) lost 60·23 grm. N. On the thirteenth day following the accident *Case* 3 had an infarction of the lung and there was an abrupt rise of body temperature coupled with a slight rise in the urinary volume and N excretion. These rises subsided after three days, but the temperature subsequently rose and clinical evidence of pneumonic consolidation was present. Unfortunately this development concluded the biochemical record. It was apparent that even with definite fever the rise in N excretion was not as great as occurred in consequence of the initial injury.

Throughout the period of observation this case was on a constant intake of water, the volume of urine varying with the N excretion.

In *Case* 4 the basal oxygen consumption and body temperature curves fluctuated in parallel but not with the N excretion. On the eighth and ninth days following the injury there were abrupt rises in both the former curves although the N excretion was decreasing at the time. Subsequently there was a slight rise in N excretion, but this took place some four days later and may not have been related to the former events. A rise in body temperature and basal oxygen consumption is not necessarily accompanied by a rise in N excretion.

The Effect of Supplementing Diets of Average First-class Protein with Certain Amino Acids (*Cases* 5–7).—On the supposition that in these cases of injury the organism might require to catabolize its reserves to supply some essential amino acid in greater amount than was normally supplied in the diet—a condition analogous perhaps to the catabolism of body proteins in birds following the moulting period—1 grm. *l*-cystine was added daily to the low protein (mainly first-class) diet of *Case* 5. As the result of a fracture of both bones of the leg at the mid point this man lost 151·78 grm. N in twelve days. The urinary volume varied with the urinary N. Although the oxygen consumption rose with the rising urinary N output, it fell during the fourth to sixth days at a time when the N output continued to remain high. The rise in body temperature preceded that of N output and oxygen intake. The maximum temperature, 38·22° C., occurred on the second day.

To determine if there were any changes in the excretion of Na and K, the period of fifteen days was divided into three lots of four days each and one group of three days, and the average daily excretion of these elements determined for the various periods. K rose from 2·30 to 4·83 grm. per day during the third period—corresponding to the maximum N excretion—descending during the fourth period to 4·39 grm. Na, on the other hand, fell slightly during the second period, rising to the initial value by the fourth period.

The level of K in the blood serum fell slightly during this period. On the second, fifth, and eighteenth days its values were 23·1, 22·9, and 21·4 mgrm. per 100 ml. respectively. On these occasions the serum Na levels were 314, 348, and 294 mgrm. per 100 ml. These blood serum values are fairly constant: such variations as may appear significant are opposite in kind to those occurring in the urine. The corresponding blood urea N values were 23, 17, and 16 mgrm. per 100 ml., those of serum S 2·4, 0·9, and 0·8 mgrm. per 100 ml., indicating a definite initial rise in the level of these blood constituents, a rise which soon declined to normal.

As it has recently been suggested that glycine may play a part in the restoration to normal of the partially atrophied muscle fibres in cases of muscular dystrophy (literature reviewed by Cuthbertson and Maclachlan, 1934) it was thought of interest to feed glycine to one or two cases of traumatic injury.

Case 6, whose tendo Achillis had been severed by a reaping machine, received 10 grm. glycine daily throughout most of the period of observation in addition to a diet of average first-class protein content. In the course of ten days 14·70 mgrm. N were lost. The urinary volume varied with its N content but tended to rise still further. Body temperature also varied to some extent with urinary N.

Case 7, who had been knocked down by a motor-car with consequent fracture of both bones of one leg and considerable bruising, lost 91·23 grm. N in the space of nine days despite the addition of 15 grm. glycine daily to a diet of average first-class protein content. The tibia was fractured at the junction of the proximal and second quarters; the fibula at its head.

The urinary volume, urinary N, and body temperature tended to vary in parallel as occurred in the previous case.

As the process of testing out each amino acid in turn appeared to be wasteful without some positive indication, it was resolved to test out some palatable hydrolysate of mixed animal tissues.

Effect of Addition of a Hydrolysate of Mixed Ox Tissue (*Case* 8).—A palatable hydrolysate of mixed ox tissues was found in the preparation Eatan. About 71 per cent of the total N of this proprietary product is reported to be combined in amino acid form. To the rather low first-class protein diet of *Case* 8 were added daily 50 ml. (2·53 grm. N) of this mixture. The patient—a footballer with both bones of one leg fractured at the junction of the upper and middle thirds as the result of kicking an opponent's boot—lost 112·03 grm. N in the course of eighteen days. The urinary volume fluctuated with its content of N. During the gradual rise in N output there were periodic rises in body temperature, but these had passed off and the temperature had become normal before the maximum daily output of N was reached.

Since movement, certain meat extractives, and amino acids had all failed to stem the loss of body N in these injured men, it was thought that some measure of evidence might be achieved by adding certain incomplete proteins in large amount to the diets of injured persons.

Effect of Addition of Gelatin (up to 200 grm. daily) to Diets of Low First-class Protein Content (*Cases* 9, 10).—*Case* 9 fractured his right tibia at the mid-point as the result of indirect violence, there being no apparent displacement of the bone-ends. From the third to seventh days he received daily 120 grm. gelatin and then 150 grm. daily for six days. The basal diet was low in protein (mainly second-class) and normal as regards carbohydrate and fat. The patient lost 64·60 grm. N in ten days. Urinary N and volume ran parallel. When the gelatin was finally omitted from the dietary the urinary volume did not decline proportionally. The body temperature changes paralleled the N excretion. Preformed creatinine initially varied with the urinary volume and urinary N excretion, but later did not fluctuate with these changes, tending to remain fairly constant. The creatinuria was almost as great as the excretion of preformed creatinine. Increment in gelatin intake led to an increase in creatine excretion and decrease in intake led to a diminution in the creatinuria. This point will be discussed later.

Case 10, whose injury consisted of the fracture of both bones of one leg, at the mid-point, the result of a kick on the football field, received 200 grm. gelatin daily throughout the period of observation. From the sixteenth day following the injury it was omitted. From the second to sixteenth days the patient lost 15·30 grm. N. The urinary N varied with the total volume of urine passed during the gelatin feeding period, but the body temperature was never above 36·83° C. during the whole period of observation. The preformed creatinine excretion was very constant. The creatinuria was marked and its daily fluctuations corresponded with the changes in total urinary N. On ceasing to give gelatin it quickly declined, and within three days only a trace was present.

The most marked creatine excretions of the series were noted in these two cases and in *Case* 6, who received 10 grm. glycine daily. The degree of creatinuria in the former two cases appeared to be definitely related to the ingestion of gelatin, and since glycine itself appeared to produce a similar effect it may be reasonable to assume that the increased creatinuria induced by gelatin feeding was related to its high glycine content.

Many years before, Gibson and Martin (1921) had noted that the administration of gelatin to cases of muscular dystrophy led to a creatinuria. These observations in injured subjects suggest that their muscular function is deranged in some peculiar way. It has long been known that fracture cases frequently exhibit a creatinuria.

The Effect of Sodium Caseinate (175–200 grm. 'Protosol' daily) when added to Diets of Very Low First-class Protein Content (*Cases* 11–17).—*Case* 11 received his injuries—a fractured humerus at the junction of the upper

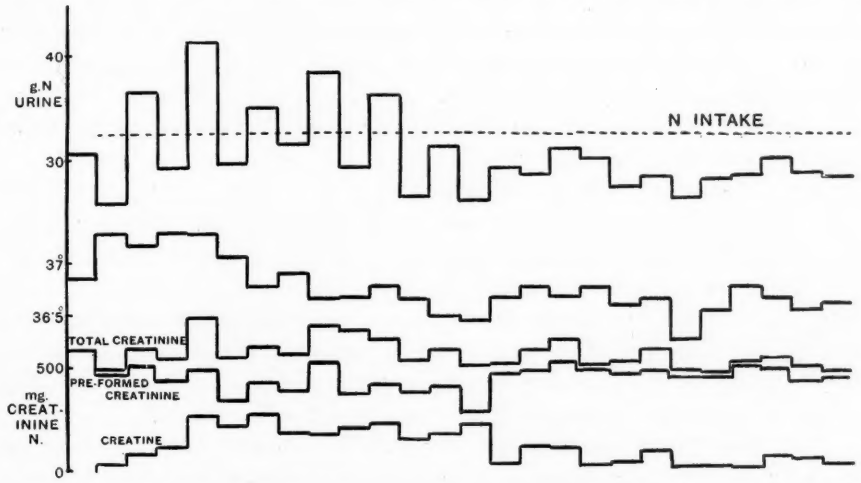

FIG. 342.—*Case* 11. Daily fluctuations in average body temperature, urinary N, and total creatinine, preformed creatinine, and creatine (expressed as creatinine N).

and middle thirds, with bruising plus a lacerated wound over the right supra-orbital ridge—as the result of colliding into a wall when bicycling. His arm was massaged daily and gently moved before being put back into the splint. For the first week he stayed most of the time in bed as his body temperature was slightly above 37° C. for some six days. The patient lost 13·75 grm. in twenty-three days. The main loss of substance took place during the first ten days, when

39·59 grm. N were excreted in excess of intake, and from the fifteenth to twenty-seventh days 25·84 grm. N were retained. In addition to a diet low in first-class protein he consumed 175 grm. 'Protosol' daily.

The urinary volume fluctuated with the N excretion, but later continued to rise as the N gradually fell. The urinary N and the body temperature curves were parallel, the daily fluctuations corresponding (*Fig.* 342). Preformed creatinine fluctuated with the urinary changes, but as the urinary N rose the preformed creatinine decreased slightly, rising again as the urinary N fell. Creatine excretion rose from zero, then remained parallel to the total urinary N, and finally declined as the latter fell, becoming eventually little more than a trace. The degree of creatinuria is apparently related to the total protein catabolism, and this experiment would suggest that as this creatinuria waxes the preformed creatinine output wanes slightly.

The basal diets of *Cases* 11 and 12 were creatine- and creatinine-free.

Case 12 received 200 grm. sodium caseinate daily during his convalescence from the fracture of both bones of one leg at the mid-point caused by a direct blow by a machine handle. The wound, which was compound, was incised and cleaned under chloroform and ether anæsthesia. No sepsis resulted. During nine days the patient lost 50·88 grm. N. The urinary volume rose with increase in N excretion and continued rising even after the N had reached its maximum value. The maximum body temperature (37·44° C.) coincided with the maximum N excretion. Preformed creatinine remained fairly constant, though as in the previous case a tendency to fall as the creatinuria developed was noted. The latter initially varied with the total N excretion, but later fell although the N output still remained high.

Case 13 had the lateral and medial malleoli of the bones of one leg fractured as the result of a wagon passing over his leg just above the ankle. Though the diet contained 23·82 grm. N daily, the patient during seven days of observation lost 36·79 grm. N. Total urinary S fluctuated in parallel with total N and urinary volume. As no precaution had been taken to prevent S loss in the fæces during drying, only the excess outputs of N and S in the urine can be compared. On this basis the S : N ratio of the material lost was 1 : 16·36. The S : N ratios of intake and total urinary outputs were 1 : 13·65 and 1 : 13·79 respectively. The S : N of the urinary excretions during the first and second periods of five days were 1 : 13·63 and 1 : 13·94 respectively. The excretion of preformed creatinine rose slightly, the rise corresponding to the rise in total N. The creatinuria diminished slightly. Owing to the fact that the patient was on a meat-containing diet little value can be attached to this apparent reciprocal relationship.

In this case there was practically no disturbance of body temperature.

Case 14 received a compound fracture of both bones of one leg (fracture at the mid-point) as the result of having his leg crushed between two large drain pipes. During the period of observation (thirteen days) he lost 27·26 grm. N, the daily intake being 26·57 grm. The excretion of total S in the urine varied with that of total N and the volume of urine passed showed similar changes though occurring somewhat later.

The maximum values for the oxygen consumption, body temperature, and pulse-rate coincided in time with the maximum excretions of N and S in the urine, and the trend of all these values was in the same general direction—

namely, an irregular rise to a maximum on the seventh day and an irregular fall thereafter. The respiratory quotient varied between 0·76 and 0·85 during first twelve days ; thereafter it gradually rose to 1·15. The output of preformed creatinine was fairly constant; such variations as occurred were parallel to the general trend of N and S. The degree of creatinuria was slight and remained practically constant throughout the period of observation.

The fall of a coal tree fractured both bones of both legs of *Case* 15. Both bones of the right leg were fractured about the middle, those of the left leg at the junction of the proximal and second quarters. During the ten days of observation he lost 17·59 grm. N even though on a diet of high first-class protein content

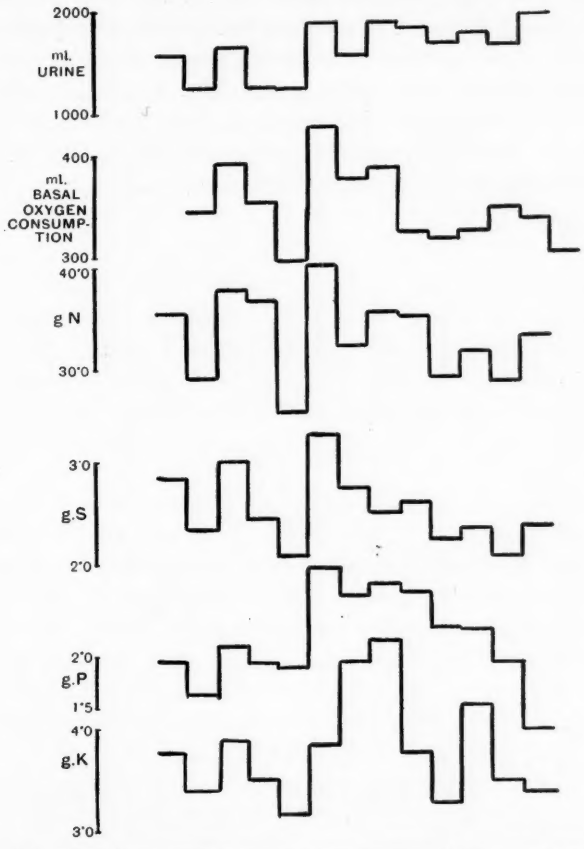

FIG. 343.—*Case* 15. Daily basal oxygen consumption, volume of urine, and urinary output of N, S, P, and K, following the fracture of both bones of both legs.

(31·84 grm. N daily). The urinary volume, total N, total S, total P, and to slightly less extent K, exhibited similar daily variations (*Fig.* 343). The values did not show the characteristic slow rise to a maximum and subsequent gradual fall noted in most of the other cases. The daily fluctuations in the excretion of these catabolities were paralleled by similar fluctuations in the basal consumption of oxygen, body temperature, and pulse-rate, indicating that these irregularities were not simply variations in the daily excretion but due to differences in the rate of metabolism. The excretion of preformed creatinine remained remarkably constant;

such fluctuations as occurred corresponded with those of the other urinary catabolities. The creatinuria gradually rose to a maximum on the eleventh day following the injury, then declined.

As the result of being charged by an opponent when playing football *Case* 16 fell heavily to the ground, both bones of one leg being splintered at their mid-point. The leg was so swollen on admission to hospital that it was impossible to put it in a splint. For five days it lay between sand-bags. It was then manipulated and splinted. Six days later a pin had to be passed through the os calcis and extension applied. The leg was still considerably swollen at the site of injury sixteen days after the accident. During these two days of manipulation the intake of food was greatly diminished, 5 grm. N being consumed on the first occasion and less than 2 grm. on the second occasion. The patient lost 45·82 grm. N in the course of fifteen days. The average daily intake of N inclusive of the two days of manipulation was 33·84 grm. The surgical procedures each produced a definite but temporary increased excretion of N over and above the general rise and fall resulting from the original injury. The urinary volume and body temperature varied with the total N excretion.

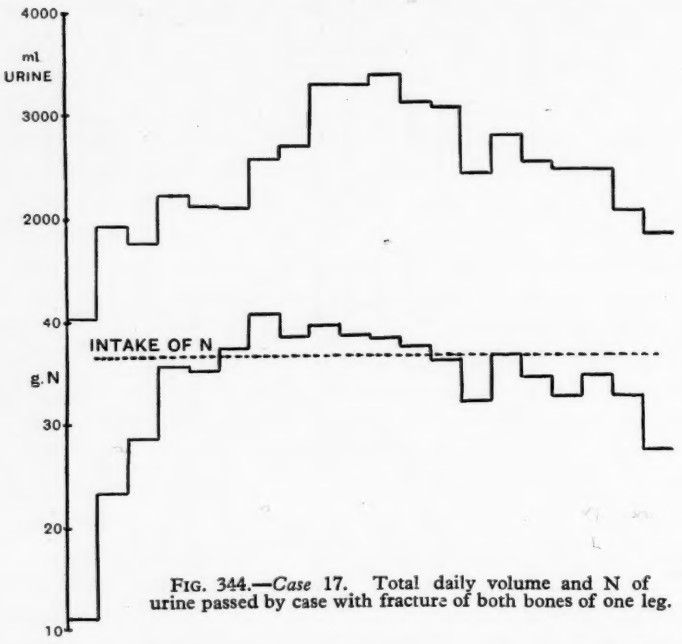

FIG. 344.—*Case* 17. Total daily volume and N of urine passed by case with fracture of both bones of one leg.

Case 17 (*Fig.* 344), the last member in this group receiving high protein diets, sustained a fracture of both bones of one leg just above the ankle due to a fall of stone. The daily intake of N was 36·69 grm. (meat, eggs, and milk mainly). During the seventeen days of observation the patient stored 25·49 grm. N.

From this experiment it appears that on superimposing a diet containing the maximum amount of first-class protein which can be ingested over a period with a minimum of discomfort, upon a diet of average N content, there occurs first a period of retention while the N excretion is rising, then a period of loss, to be followed later by another period of retention. The fact that there was a loss of N during a considerable part of the period appears to indicate that in such cases

of injury attempts to stem completely the loss of body tissue fail during the period of maximum tissue catabolism.

The maximum volume of urine was passed subsequent to that of the total N.

This group of experiments indicates that in cases of fracture due to direct violence, where in addition to the breaking of bone there is a definite amount of soft tissue damage (mainly muscle) with presumably extravasation of blood, attempts to maintain these patients in nitrogen equilibrium during the period of maximum catabolism fail. The loss is less the higher the protein intake.

The caloric value of the last group of cases (*Cases* 13–17) varied between 3044 and 3606 Cals. The recorded N losses in all the cases are slightly greater than the actual, since no account has been taken of the sweat N.

The Effects of Diets of High Carbohydrate and High Calorie Content (*Cases* 18, 19).—In consideration of the sparing action of carbohydrate on the rate of protein catabolism *Cases* 18 and 19 were fed with mixed diets of high carbohydrate content—diets of 4135 and 3592 Cals. respectively.

Case 18 had the lower end of the left fibula fractured as the result of a fall of stone. There was practically no displacement of the fragments. In the course of nine days this man lost 35·90 grm. N (daily intake 16·39 grm. N).

Case 19 suffered from a fracture of the lower ends of both bones of one leg. The ends were manipulated into position without anæsthesia. During the course of five days the patient lost 47·57 grm. N (daily intake 8·31 grm. N).

In both cases the urinary volume varied with the excretion of N. The body temperatures did not rise concurrently with the total N. In *Case* 19 the maximum temperature (37° C.) was recorded three days after the maximum N excretion.

DISCUSSION

In the course of the foregoing experiments attempts have been made to prevent the loss of N which occurs during the period of increased catabolism that follows severe injuries due to direct violence. In the light of the data derived from patients receiving diets rich in first-class protein and of high caloric value it is obvious that no success could have attended the earlier experiments where supplements of meat extractives, certain amino acids, and incomplete proteins were given to subjects receiving diets of average or low caloric value and low in first-class protein.

From certain incomplete observations on cases of fracture due to indirect violence where little or no tearing of soft tissue had resulted from the displacement of the bone fragments, the evidence suggested that N equilibrium could be attained more easily, particularly when the cases received similar mixed diets of high protein content.

Reference to the early literature indicates that when high protein diets or low protein diets of very high caloric value are fed to normal uninjured persons, N retention may apparently occur. Krug, a pupil of v. Noorden's (q.v.), carried out experiments on himself, a healthy individual showing no sign of wasting. During a preparatory period of six days he attained N equilibrium on a diet of 2590 Cals. (44 Cals. per kilo.). On raising his intake by an additional 1700 non-protein Cals. he retained 49·5 grm. N during fifteen days, the retention being as great at the beginning as at the end. Bornstein (1898) noted a retention of 16 grm. N in fourteen days when he supplemented a diet sufficient to maintain N equilibrium with an additional 40 grm. protein. The most outstanding example of this

storage phenomenon has been described by Lüthje (1902), who noted gains of 10 to 20 grm. N daily in a subject in good condition, who consumed a daily diet of 6000 Cals. containing 380 grm. protein. C. Voit had shown that the increased amount of flesh that is put on by the dog during a surplus diet is frequently lost on return to the normal diet. Dengler and Mayer (v. Noorden, 1909) found that a man aged 42, previously in a good state of nutrition, gained 371 grm. N in seventy-two days when the diet was gradually raised from 2183 to 4695 Cals. and then reduced. The maximum gain occurred during the period of maximum caloric intake.

In order to test this apparent capacity of the normal human organism to store N when placed on a high protein diet, the retentive powers of two healthy male students, H. M. and J. H., aged 19 and 23 years respectively, were examined.

Having attained N equilibrium on a well-balanced normal diet containing 16·78 grm. N, student H. M. was placed for six days on a diet of similar caloric value but containing double that amount of protein—mainly first-class (33·75 grm. N; 2822 Cals.).

	Grm.
Daily basal intake N	16·78
Daily urinary N during basal intake	14·90
Daily fæcal N	1·49
Total daily output on basal	16·39
Balance (excluding loss of N by sweat)	+ 0·39
Extra food N (6 days) over basal	101·82
Extra urinary N over basal	100·18
Balance	+1·64

The retention of 1·64 grm. N as the result of six days' excessive N feeding is insignificant, as it is probable that some extra N would have been excreted in the fæces and sweat during the period of high protein intake.

The second experiment (student J. H.) was unsatisfactory in many aspects, but does indicate a similar finding.

	Grm.
Basal food N (10 days)	145·7
High N diet (10 days)	324·3
Extra food N (10 days)	178·6
Extra urinary N over basal	179·0
Balance	−0·4

This figure (−0·4 grm.) is negligible.

It is apparent that these two individuals did not exhibit any capacity for retaining N when placed on a high protein diet such as the fracture cases received. The writer is therefore unwilling to believe that the retention phenomena described by earlier workers is a general occurrence, though undoubtedly certain people increase in weight when their intake of food is increased, and, though this increase in weight is mainly fat, it is possible that protein may also be stored as indicated by N retention.*

* Since this was written further experiments on student H. M. and on the writer himself indicate that when first-class protein (in the form of milk) is added to a diet adequate in calories but low in first-class protein (80 grm. protein: 23 grm. first-class) N retention occurs.

The actual fracture of bone probably causes but little disturbance, the major disturbance apparently resulting from the tearing of the soft tissues by the displaced bone fragments and from the damage done to the soft tissues by the traumatic agent itself. There appears to be no obvious relationship between the amount of damage and the catabolic response. Individuals react differently. The most severe injuries of this series were those of *Case* 15 (fracture of both bones of both legs). In this case the loss was only 17·59 grm. in ten days, the N intake being 31·84 grm. daily.

According to Linser and Schmid (1904), no breakdown of tissue protein occurs in normal persons as the result of the external application of heat if the body temperature is maintained under 39° C. Graham and Poulton (1912–13) noted no appreciable increase in N excretion as the result of raising the body temperature to 39·44° to 40° C. by exposure to steam heat. It is unlikely, therefore, that an appreciable part of the increase in catabolism observed in these injured subjects is due to the slight pyrexia sometimes found. It is much more reasonable to believe that the increase in temperature is (in part at least) due to the increased catabolism.

Absorption of digestion products from the gut cannot be appreciably affected in injury, as in only one case (*Case* 17, who received maximum protein) did the fæcal N excretion exceed an average of 2·0 grm. per day.

Several attempts have been made in the past to maintain N equilibrium at the height of an infective fever. Shaffer and Coleman (1915) believed that the lack of success lay in the fact that food of insufficient fuel value and of unsuitable character had been used. The work of Lusk (1891), Sivén (1901), Landergren (1903), Folin (1903), Murlin (1907), and others had shown the superior value of carbohydrate in sparing body protein in fever. Shaffer and Coleman were, however, able in only one instance to obtain N equilibrium in the early stages of typhoid and they were unable at any time to maintain it for more than two to three days when the temperature was above 38·89° C., although the patients were receiving as much as 90 Cals. and 1·6 grm. protein per kilo. per day. Rolland (1912) almost attained N equilibrium in febrile patients with diets high in both calories and protein, but Coleman and Du Bois (1915) failed to establish N equilibrium in cases of typhoid and other fevers on diets containing adequate calories and as much as 15 grm. N daily. Lauter and Jenke (1925) and others have also failed to establish N equilibrium in fever with diets of high caloric content.

The increased N excretion in febrile infections is due not only to the extra liberation of energy but also to an active destruction or autolysis of tissue. The failure to attain N equilibrium at the height of catabolism in the present series of cases is all the more extraordinary therefore, considering that there was little or no disturbance of body temperature.

In those subjects on whom basal metabolic determinations were made the oxygen consumption varied with the body temperature and pulse-rate, and in most cases this corresponded to the maximum period of N excretion. This period of maximum excretion was reached from the fourth to the eleventh day following the injury, generally the sixth day in this series. The amount of N excreted generally rose gradually to a maximum and then slowly declined.

The excretion of total urinary S ran parallel to the curve of N excretion in

the two cases (*Cases* 13 and 14) in which it was followed throughout the experimental period. Relative to N slightly more S was excreted in the first half of the experimental periods, which lasted ten to sixteen days respectively. The S : N ratio of the material lost by *Case* 13 was 1 : 16·36, a ratio which indicates the catabolism of muscle substance.

Although balances were not struck in the case of P, K, and Na, it was apparent that the excretion of the former two elements varied with that of N, while Na remained fairly constant. The fact that K and not Na was the variable element suggests that cellular material was being catabolized.

The fluctuations in the urinary volume were mainly dependent on variations in the rate of excretion of the products of catabolism.

Omitting for the moment those cases receiving gelatin or creatine- and creatinine-containing foods, the two cases remaining (*Cases* 11 and 12) in whom the creatine and creatinine excretions were studied exhibited creatinurias paralleling the changes in urinary N. The creatinine excretion tended to vary inversely with the degree of creatinuria. This was more noticeable in *Case* 11. The combined creatine and creatinine excretion rose and fell with the total N excretion, the fluctuations being less marked in the case of creatine alone. These increases were not due to a washing out of catabolites, for the urinary volume rose to its highest level during the period of total N and total creatinine decline. These observations suggest either that the metabolic process which has creatinine as an end product is in part arrested at the stage of creatine, or that these two metabolites have separate origins, the creatinuria arising from the breakdown of perhaps local muscular tissue, the diminution of creatinine excretion being the result of a general decline in the activity of the total protoplasmic mass following on the injury. The latter is perhaps the more reasonable explanation, but if true it would indicate that the main catabolism is local and not general, a conclusion somewhat difficult to believe in the face of the marked loss of N which may sometimes occur—137 grm. in ten days, but yet supported by clinical evidence of localized atrophy.

In those cases receiving gelatin (*Cases* 9 and 10) the excretion of preformed creatinine was fairly constant and independent of the amount consumed. On the other hand, the creatinuria was dependent in part on changes in the intake of this protein, for, on increasing or decreasing the amount taken, similar changes occurred in the amount of creatine excreted. The level of the creatinuria in these two cases and in those receiving glycine was the highest of the series, though never exceeding 650 mgrm. (in terms of creatinine N), and, in those cases receiving creatine- and creatinine-containing foods (*Cases* 5, 14, and 15), the degree of creatinuria was often very constant and generally of a lower value than was observed in the other cases.

So far it has only been possible to trace the fate of the nitrogen and sulphur residues of the protein which has been catabolized.

No account has been taken of the deaminized residue or of the contemporaneous catabolism of carbohydrate and fat reserves. If oxidation of the substances is in excess of normal, then the energy liberated must appear as heat or work. That there is an increase in the oxidative processes of the body is certain, and that the slight rise of body temperature is not a sufficient explanation of the manner in which the liberated energy is expended is also certain. Of work done we have no knowledge except that the ordinary processes of healing will require a greater

expenditure of energy than normal for the synthesis of new tissue and for the removal of effete material. Therefore, although this excessive catabolism which follows severe injury caused by direct violence persists in the face of what is probably the maximum protein and caloric intake that the human organism can consume when confined to bed, it is modified to a considerable extent by such diets. A somewhat similar dietary effect has been noted in the case of the infective fevers. Whether the primary demand is for essential amino acids to repair the damaged zone or for fuel, it is impossible to state accurately; probably both are required, for in the two cases (*Cases* 18 and 19) which received diets of high caloric value but of average or low protein content little success was achieved.

Disuse atrophy, though a contributory factor, does not form an adequate explanation of this strange phenomenon, as the loss of body substance is greatly in excess of that produced by experimental disuse (Cuthbertson, 1929). It has long been known, however, that muscular wasting associated with bone or joint disuse is much more rapid and extreme than that which occurs with simple disuse of a limb. It is also true that there is a more rapid and extreme wasting in limbs immobilized for some inflammatory or traumatic lesion than in limbs immobilized to correct a deformity. The reason for this lies not in the degree of immobilization but in a reflex trophic effect affecting certain groups of muscles more than others in the same region. Such reflex atrophy is dependent on the integrity of the afferent nervous paths (Harding, 1925, 1926, and 1929).

One is tempted to speculate that herein may lie an explanation for this trophic change, the resultant rapid wasting of muscle being the response to the organism's urgent demand for material for maintenance and repair. As was suggested in an earlier paper (Cuthbertson, 1930) injury to an animal generally leads to lessened activity and a lessened activity to a diminished food supply. The necessity is urgent and the body may require and may prefer to catabolize its reserves to meet the exigencies of the moment. These experiments offer no direct evidence whether the wasting of the specific groups of muscles has in any way been mitigated by diet. Such may be the case. On the other hand, the local loss of substance may have proceeded unabated by such dietary measures, though to a great extent masked by anabolic processes proceeding elsewhere. Further experiments are required to elucidate this very interesting point.

Be that as it may, the beneficial effect of a diet of high protein and caloric content in diminishing this wastage of flesh that generally follows injury due to direct violence is sufficient to warrant the provision of such diets to injured patients. Accident cases can generally tolerate and even enjoy such diets and we have not found that they retard the healing of bone. It is probably advisable to supplement such high protein diets with a certain amount of base contributing foodstuffs—viz., vegetables and fruit.

SUMMARY

1. The ingestion of diets very rich in first-class protein and of high caloric value by persons suffering from the fracture of one or more of their long bones as the result of direct violence, modifies considerably the marked loss of body protein which normally occurs under such circumstances. At the height of the catabolic disturbance, however, such diets still fail to prevent this loss of protein.

2. Measures such as massage and manipulation, the addition of meat extractives, glycine, hydrolysate of mixed ox tissue, gelatin, and sodium caseinate, and diets of high caloric value but average protein content, similarly failed to stem the loss of protein and generally proved less successful in mitigating the drain on the body's reserves.

3. The catabolic disturbance is characterized by an increase in the basal consumption of oxygen with attendant rise in pulse-rate and temperature, and by parallel rises in the urinary output of N, S, P, and to a less extent K.

4. The creatinuria which develops and parallels the rise in total N is accompanied by little change in the creatinine excretion; such change as occurred took the form of a slight diminution during the period of maximum creatinuria.

5. Two control subjects who received diets rich in first-class protein and of high caloric value exhibited N equilibrium.

6. It is recommended that injured patients should receive diets of high protein and caloric content.

In conclusion I wish to express my thanks to Professor E. P. Cathcart, F.R.S., for his helpful criticism, to my clinical colleagues of the Royal Infirmary, Glasgow, for their readiness to supply me with suitable patients, and to Miss Cumming for her unremitting care in the conduct of these experiments. I also wish to thank the two students H. M. and J. H. who acted as control subjects. I am indebted to the Medical Research Council and to the D. C. Andrew Research Fund for grants in aid of this research.

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ŒSOPHAGECTOMY FOR CARCINOMA OF THE THORACIC ŒSOPHAGUS

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Of all the conditions that are difficult of treatment because of their nature or inaccessibility, carcinoma of the thoracic œsophagus has seemed to be one of the least amenable to successful surgical attack. As Grey Turner[11] has said, it "has usually been considered the most unsatisfactory subject in the whole realm of surgery". Nevertheless in the last few years a number of cases successfully treated* have been reported by Torek,[10] Lilienthal,[9] Eggers,[1, 2] and Grey Turner.[12] The pioneers, Mickulicz, Richter, Quénu and Hartmann, Rehn, Enderlen, and Sauerbruch amongst others, whose work undoubtedly paved the way for the subsequent successes, deserve more than a passing thought for a courage, determination, and enterprise that is seldom required of modern surgeons. Nevertheless success does capture the imagination, and Torek's[10] transpleural œsophagectomy in 1913 will remain one of the outstanding feats of surgery. Since then Eggers[1, 2] has had, by this method, two successful cases. Lilienthal[9] performed an œsophagectomy by the posterior mediastinal route. By a modification of a method unsuccessfully used by Teutonic surgeons,[4, 8] Grey Turner[11, 12] recently has had a successful case.

A number of successful operations for carcinoma of the lower end of the œsophagus or the cardia of the stomach, and for non-malignant tumours, have been recorded (Gohrbandt,[5] Hedblom,[6] Krauss,[7] Voelcker,[13] Zaaijer,[14] amongst others), but these need not be discussed here.

The following is the report of a case in which a satisfactory result has been obtained.

CASE REPORT

Mrs. B. K., aged 56 years, had had dysphagia for eight months. This had been worse for the last two months, so that for two or three weeks she had been able to swallow only fluids. There had been no pain in the chest.

X-ray examination (barium swallow) revealed an obstruction of the œsophagus opposite the junction of the sixth and seventh thoracic vertebræ (*Figs.* 345, 346). Œsophagoscopy, performed by Dr. A. Blaubaum, revealed a mass projecting into the œsophagus and constricting it, 12 in. from the incisor teeth. A portion of the mass was removed, and on microscopical examination this showed an epidermoid carcinoma.

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* 'Success' must of necessity be a relative term when dealing with malignancy. Torek's case is actually the only one in which the disease was completely eradicated, his patient living for thirteen years. However, the operative successes give promise of hope for cases which are diagnosed sufficiently early.

The patient and her relatives were informed of the diagnosis and of the risks of surgical intervention and were agreeable for any treatment to be adopted. She was admitted to the Royal Melbourne Hospital on Feb. 21, 1935. Her general condition was good except that there was pyorrhœa round some of her lower teeth. Her renal function was satisfactory and the lungs were clear. The Wassermann reaction was negative. Her weight was 7 st. 6 lb., about a stone below her normal weight.

Gastrostomy (Senn) was performed under local block anæsthesia on Feb. 22. Convalescence was satisfactory except that, on the day following operation, the patient vomited, apparently as the result of a morphia idiosyncrasy. Omnopon was used during the remainder of her stay in hospital.

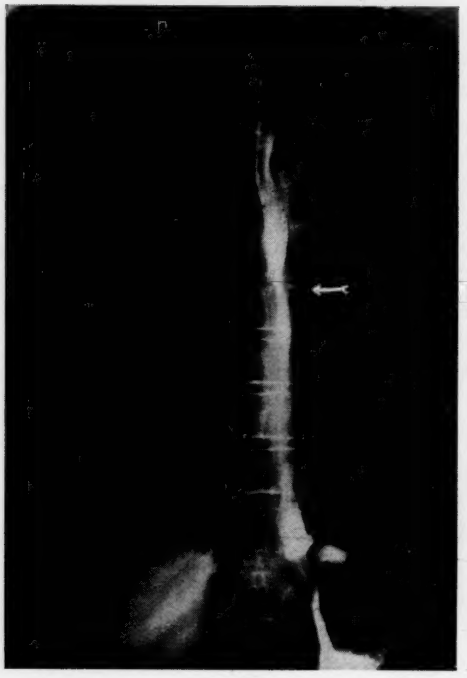

FIG. 345.—X-ray photograph of the chest showing the obstruction in the œsophagus opposite the junction of the sixth and seventh thoracic vertebræ.

FIG. 346.—Obstruction to the œsophagus shown in oblique view after administration of a thin meal.

Energetic treatment of the mouth condition had been adopted, though the teeth were not removed. She was given a high caloric diet, with adequate vitamin content, through the gastrostomy tube (three-hourly feeds). Artificial pneumothorax of the left side was produced by injection of increasing amounts of air on each second day.

Five days later (Feb. 27) under gas and oxygen anæsthesia, through a paravertebral incision, the vertebral ends of the 4th, 5th, and 6th ribs on the left side were cut and the intercostal vessels and nerves were ligated and cut. The wound was then sutured. The patient recovered from this procedure uneventfully and as her general condition was good it was decided to proceed with the œsophagectomy.

Three days later (March 2) œsophagectomy was performed. Under avertin and intratracheal gas and oxygen anæsthesia, with the patient in the right lateral position, an incision was made along the sixth intercostal space, this incision connecting at its vertebral extremity with the lower end of the paravertebral incision previously made. The muscles (trapezius and latissimus dorsi) were incised and hæmostasis obtained. The wound made at the preliminary operation in the trapezius and rhomboids, on the medial aspect of the scapula, was opened up. The intercostal muscles of the sixth space and the underlying pleura were now incised and the incision in the pleura continued upwards, at the vertebral end, to the 3rd rib.

A rib-spreader was introduced and a clear view of the pleural cavity obtained. The lung was collapsed against the mediastinum and there were about 4 oz. of slightly blood-stained fluid in the cavity. The fluid was aspirated. The hilum of the lung was normal and no nodules could be felt in the lung tissue. One fairly dense adhesion was present between the apex of the lung and the parietal pleura. This was divided.

An incision was then made in the mediastinal pleura, just in front of the descending aorta, from the diaphragm to the arch of the aorta, and a further one, in line with this, from the arch to the cupola of the pleura.

The mediastinal tissues in the lower part were dissected, partly by sharp and partly by blunt dissection, and the œsophagus freed so that a tape could be passed round it, and separated upwards and then downwards from this point until it was free in the whole of its extent from the diaphragm to the arch. The major vessels were grasped by forceps, cut and then sealed with the electric cautery. Both vagi were cut across sharply opposite the left bronchus, at an early stage of this procedure. This did not cause any appreciable alteration in pulse-rate or blood-pressure. The tumour was found just below and partly behind the arch of the aorta and appeared not to have infiltrated the surrounding structures.

Dissection of the mediastinum above the arch of the aorta was now undertaken and the œsophagus freed down to and around the tumour. Attention was then directed again to the œsophagus below the arch. It was drawn out and moist gauze was placed in the œsophageal bed. The upper part of the stomach was drawn slightly through the diaphragmatic opening and a purse-string suture was placed in its upper part so as to surround the œsophagus. The lower end of the œsophagus was tied in two places with heavy silk and was cut across with the electric cautery. The lower end was invaginated into the stomach and the purse-string tied. A second purse-string was introduced into the diaphragm just peripheral to the first one, and also tied.

The upper cut end was covered by a rubber sheath, which was tied on by two separate ties at an interval of an inch, to prevent leakage of infected material from the cut end on to the mediastinal surface. The œsophagus was then brought round the arch of the aorta so that it hung from the upper part of the mediastinum into the pleural cavity.

The wound was then closed temporarily and an incision was made in the neck along the anterior border of the sternocleidomastoid in its lower portion. Dissection was carried posteriorly down to the œsophagus and along this into the superior mediastinum until the cervical and thoracic dissections were joined. The chest wound was then re-opened and a pair of forceps passed into the neck wound and

down into the mediastinum until it could be seen in the chest. It was thus possible to grasp the more distal tie on the œsophagus and to draw this tube into the neck.

A drainage tube was passed through the eighth intercostal space in the posterior axillary line. The chest wound was then closed. The mediastinal pleura was left unsutured, intentionally, in order to allow drainage.

The œsophagus was then inspected and the site of division was determined. An incision was made in the skin just below the clavicle, and the subcutaneous tissues were tunelled from the incision in front of the sternocleidomastoid to the lower incision. The œsophagus was brought through this tunnel, sutured to the skin edges, and the protruding portion (containing the tumour) was then removed by incision with the electric cautery. A few interrupted sutures were introduced to unite the mucosa to the skin. A small drainage tube was passed into the upper mediastinum through the main neck wound, which was then sutured. Dressings were applied and the patient returned to bed. The drainage tube in the chest was connected with a negative pressure apparatus. A transfusion of a pint of blood was given and saline administered by the subpectoral route and glucose-saline as a continuous rectal infusion.

Two hours after the operation the patient had a pulse-rate of 132. She seemed comfortable and was given injections of omnopon at intervals as required. Her temperature rose to 103° in the evening.

On the following day the patient looked remarkably well, though her respirations were somewhat laboured—jerky and grunting in type. Her pulse-rate ranged from 120 to 140, and in the evening her temperature rose to 104°. In the twenty-four hours after operation 18 oz. of fluid had drained from the left pleural space. Clinical examination, advisedly cursorily performed, showed the left lung to be expanded. There was a considerable amount of mucous blood-stained discharge from the opening of the œsophagus.

On the second day after operation the respirations were much easier. The pulse-rate varied between 120 and 130. The temperature was 102°. The drainage from the chest had been only 3 oz. in the twenty-four hours. There was less discharge from the œsophageal opening. The drainage tube was removed from the neck, and the sutures in the neck wound were removed.

On the third day the patient was progressing well. As there had been no discharge from the chest during the preceding twenty-four hours the drainage tube was removed. She was allowed to swallow water, this being caught in a basin. From this time the œsophagus remained clear (except for the normal discharge of saliva), and the terminal portion healed to the skin margin rapidly (*Fig.* 347).

Two days later a rubber tube was placed in the œsophagus and connected with the gastrostomy tube so that she was able to swallow directly into the stomach. This was greatly appreciated by the patient. The tube, however, could not be left in position for more than a few hours at a time as it caused irritation of the œsophagus (*Fig.* 348).

On the seventh day she developed signs of a pleural effusion on the right side; 10 c.c. of serous fluid were aspirated, culture of which gave no growth of organisms. The signs in the chest rapidly disappeared. X-ray examination of the chest on the ninth day showed the lungs to be clear and to fill the pleural cavities completely. The ribs which had been cut were in apposition (*Fig.* 349).

FIG. 347.—Photograph of the patient taken about three weeks after the operation showing the œsophageal stoma thoroughly healed to the skin.

FIG. 348.—Photograph of the patient showing the tube connecting the œsophageal opening to the gastrostomy tube.

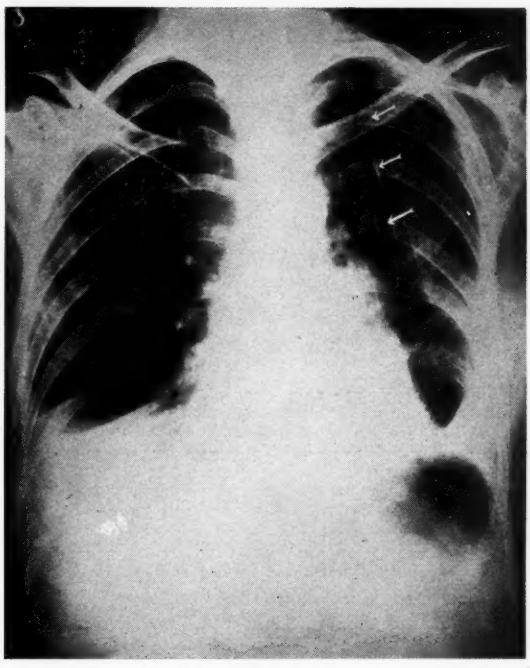

FIG. 349.—X-ray photograph of the chest of the patient taken nine days after the operation. The left lung is well expanded. There is some thickening at the site of the drainage wound. The arrows indicate the cut ribs (4th, 5th, and 6th), which are in apposition.

She was allowed out of bed on the tenth day, and from then she progressed without further complications. The wound in the chest was by this time soundly healed and the sutures were removed.

Although the general condition was improving markedly, the patient did not put on weight. It was thought that this might be due to the loss of saliva during the time when the rubber tube was not in position. A celluloid cup which had an outlet at the lower end was devised. This was placed over the œsophageal opening and the lower end connected by means of a tube with the gastrostomy. In this way she was able to obtain the greater part of the saliva escaping from the œsophageal opening. From this time she rapidly gained her normal weight.

FIG. 350.—Drawing of the œsophagus after removal. The growth is of the constricting variety with but slight superficial ulceration. The specimen, which measured just under 3 in., corresponded to about 6 in. of the œsophagus (*see* text). Note the marked wrinkling of the mucosa. The raised area running from the tumour to the upper cut edge was shown to be a leucoplakic band.

PATHOLOGICAL EXAMINATION.—Macroscopically, the specimen consists of a piece of œsophagus $2\frac{7}{8}$ in. long containing a mass in the upper (cranial) portion. The adventitia of the tube is uniform in texture and is not obviously involved by tumour.

After splitting the tube the mucosa of the lower part is seen to be normal showing a marked transverse wrinkling (*Fig.* 350). A thickening of $\frac{1}{4}$ to $\frac{1}{2}$ in. extends for $\frac{7}{8}$ in. along the wall, the upper extremity of this being within $\frac{1}{4}$ in. of the upper edge of the specimen. This cut margin appears to be normal.

A linear whitish band suggesting leukoplakia extends from the tumour up to the line of the incision.

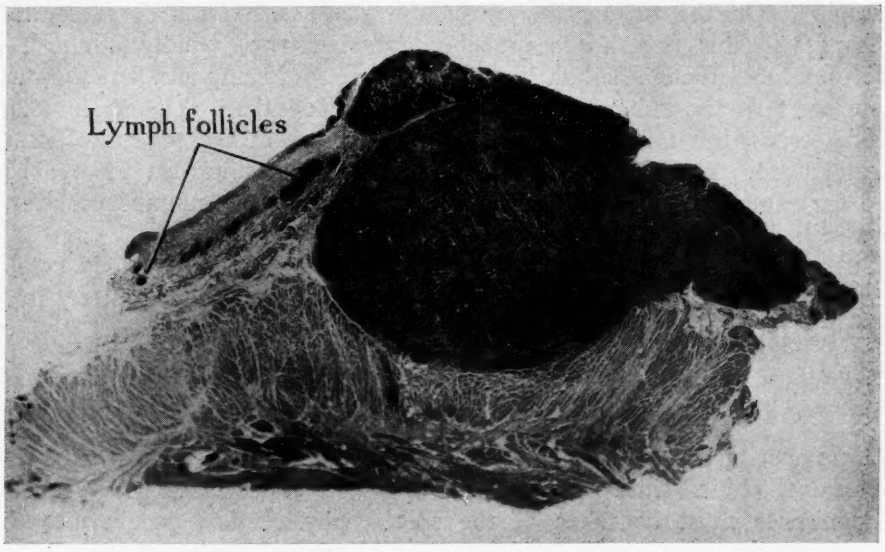

FIG. 351.—Low-power photomicrograph of the tumour, which is well circumscribed and has not invaded the adventitia of the tube. (× 4.)

(*Note.*—The actual length ($2\frac{7}{8}$ in.), as compared with the original length of œsophagus removed (about 6 in.), and the small distance ($\frac{1}{4}$ in.), as compared with 1 in. of apparently uninvolved œsophagus observed at operation, are due to the contraction of the longitudinal muscle as shown by the transverse wrinkling).

Microscopically the tumour mass is fairly circumscribed in all the sections examined, extending into the muscle coat but not into the adventitia (*Fig.* 351). The tumour is a squamous-cell carcinoma. Groups of cells showing intercellular prickles are closely arranged in a very small amount of connective tissue. In many areas the cells are large and show early keratinization (*Fig.* 352), but no definite cell nests are present. The cell nuclei show considerable irregularity in size, but only occasional mitotic figures are to be seen.

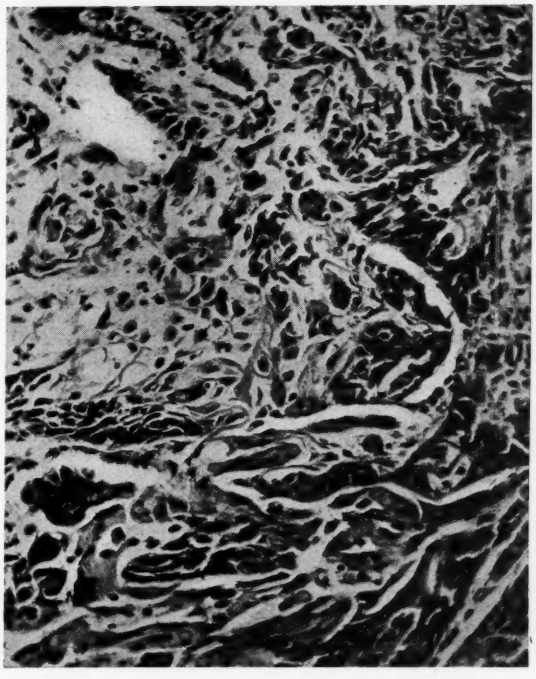

FIG. 352.—Photomicrograph of the tumour showing the epidermoid nature of the carcinoma. (× 160.)

There is a well-marked inflammatory condition in the tissues at the edge of the growth.

Above the tumour the œsophageal wall shows a marked degree of subacute œsophagitis. On the surface the epithelium shows marked hyperplasia, though the changes in this (which are probably only irritative) rapidly diminish until at the upper end the epithelium shows only some thickening and keratinization on the surface. In the submucous layer of this region there is some invasion by epithelial cords, but this extends for a short distance in a cranial direction. On the caudal side of the tumour the epithelium becomes normal more abruptly. There are some lymphoid accumulations (*Fig.* 351) in the submucous layer, but otherwise little evidence of inflammation.

SUBSEQUENT COURSE.—The patient is now very well and in good condition. There is no evidence of recurrence or metastasis. An œsophagoplasty is being undertaken and will be reported later.

DISCUSSION

A number of principles have emerged from the available mass of experimental observations, clinical investigation, and operative endeavour. These are that: (1) Cognisance must be taken of the special physiological peculiarities of the region attacked; (2) Changes brought about in organs should, where possible, be gradual; (3) The œsophagus must be removed entirely from the thorax and especially from the mediastinum; (4) Special care must be taken to avoid infection of the pleura and the mediastinum.

1. The negative pressure of the thoracic cage has always been one of the essential problems of surgery of the thorax. Many attempts have been made to overcome the difficulty by extrapleural approach to the œsophagus. As this gives inadequate exposure, the pressure chambers used by Sauerbruch were invented to overcome the difficulties. The introduction of intratracheal anæsthesia has been a further advance. Now, however, using an apparatus which delivers gas through a well-fitting mask at a slight positive pressure, even the intratracheal tube may be dispensed with.

At the end of the operation special arrangements must be made to restore the original conditions as far as possible. How satisfactory this may be is shown in *Fig.* 349, where the left lung is seen to be completely expanded and apparently normal a few days after operation. Clinical examination had shown it to be filling the chest the morning after operation. The procedure of applying slight suction automatically solves the problem of drainage of the chest, which, as shown by Eggers,[3] is an essential part of the after-treatment.

2. In transpleural approach to the mediastinum, the sudden collapse of the lung with sudden alterations in vital capacity, disturbance of blood distribution, and movements of the mediastinum, all place an unnecessary burden on an already over-strained patient. The preliminary induction of a pneumothorax overcomes these troubles.

3. Almost all attempts to anastomose the ends of the œsophagus in the mediastinum have been unsuccessful. Not only would the excision of a small piece of œsophagus be inadequate from the point of view of cure of a cancer, but this part of the alimentary canal has no cœlomic covering, and leakage is almost inevitable.

Another point which apparently is not generally appreciated is that the longitudinal muscle of the œsophagus is in a state of considerable tension. This is demonstrated by the remarkable shortening of the organ when it had been cut across. The piece of œsophagus removed in this case extended from a short distance above the diaphragm to above the arch of the aorta (at least 6 in.) and yet the specimen now measures about 3 in. (*see Fig.* 350).

It should be mentioned that anastomoses between stomach and œsophagus in the lower mediastinum have been performed.

4. The necessity for special care with regard to prevention of infection is apparent from the many observations that the majority of cases operated on die from mediastinitis or pericarditis within a few days of the procedure. Not only must all care be taken to avoid infection at the operation, but prophylaxis by thorough cleansing of the oral cavity should be undertaken. The importance of this procedure is shown (if such, indeed, is necessary) by the observation that edentulous patients may survive a perforation of the œsophagus, e.g., by foreign bodies, whereas those with pyorrhœa or other mouth infections seldom do.

It is not proposed to discuss here the relative merits of transpleural and the varous extrapleural approaches, though I am personally convinced of the superiority of the former method.

The success of this case was due, in no small measure, to the close attention to detail paid by my house surgeon, Dr. E. E. Dunlop. It is with pleasure also that I record my appreciation of advice with regard to certain technical details given me by Mr. G. Gordon-Taylor when he was in Melbourne in November, 1934.

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GASTRIC DIVERTICULA, WITH REPORT OF A CASE BEFORE AND AFTER OPERATION

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ALTHOUGH regarded as a rarity, a diverticulum of the stomach is commoner than is generally supposed. In a series of 370 consecutive opaque-meal examinations a gastric diverticulum was found in 11 cases. This series included a case of a post-operative diverticulum following a wedge excision of an ulcer on the lesser curvature. Excluding the post-operative diverticulum, so far only one of the cases has been confirmed at operation. The following is a short account of the case.

CASE REPORT

The patient, a man of 34 years, a dispenser at a London hospital, had suffered from indigestion for several years, gradually getting worse during the past two years. In character the indigestion was suggestive of a duodenal ulcer, there being marked hunger pain relieved by taking food. There had never been vomiting, hæmatemesis, or melæna. After self-treatment for some months with various alkalis, the patient eventually consulted the resident assistant physician. Physical examination was negative and the patient was referred for X-ray examination for duodenal ulcer.

X-RAY REPORT (*Figs.* 353, 354).—There is a pouch-shaped projection from the posterior wall of the stomach in the neighbourhood of the cardiac opening. The pouch is connected to the stomach by a stalk, passing into which are mucosal folds. These folds are continuous with the mucosal folds of the stomach. The pouch is situated under cover of the ribs and is not accessible to palpation. A large residue remains in the pouch after the stomach is empty and the residue persists in the pouch for twelve days. There is no ulcer niche in the stomach or duodenum and there is no other lesion detected in the remainder of the alimentary tract.

FIG. 353.—Residue present in diverticulum twelve days after meal. None of the second opaque meal, given in the erect position, has entered the diverticulum.

DIAGNOSIS.—Gastric diverticulum.

PRE-OPERATIVE TREATMENT.—The patient was kept in bed for a week. The diet was restricted mostly to fluids, and large quantities of glucose were given.

OPERATION (G. A. Ewart).—The abdomen was opened through the left upper rectus muscle. On examination it was found that the liver and stomach were rather low in position, the latter being contracted and free of gas—probably the result of the careful preparation of the patient during the previous week.

Examination of the stomach and duodenum showed no sign of ulcer. The method of approach had been previously discussed with Sir Thomas Dunhill, whom I have to thank for his help. His suggestion was to divide the 6th and 7th costal cartilages near the sternum if additional exposure was required. This, however, did not seem necessary owing to the relatively low position of the stomach. An incision was next made in the gastro-hepatic omentum and vessels were ligatured.

The lesser sac of peritoneum proved to be almost obliterated by fine filamentous adhesions. Palpation in the supposed position of the diverticulum failed to give any indication of its presence. After a tedious and somewhat anxious period of gauze dissection, the fundus

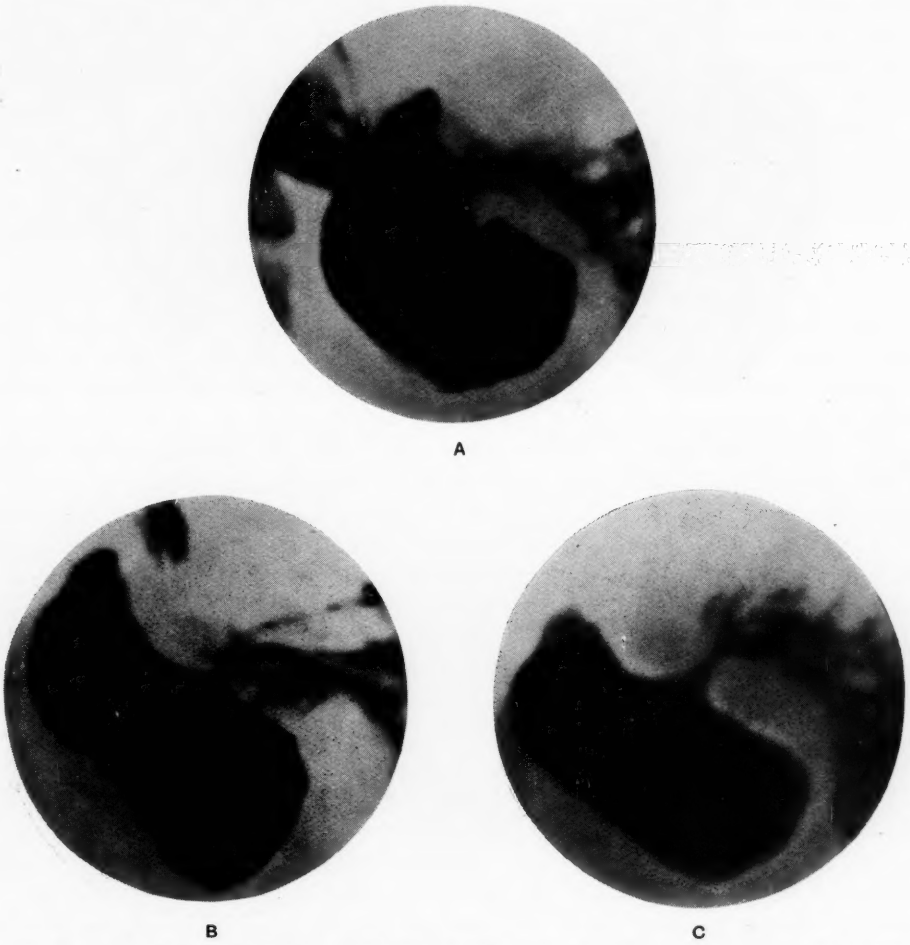

FIG. 354.—A series of aimed exposures showing the diverticulum: A, Postero-anterior view; B, Oblique view, showing mucosal folds entering stalk; C, Lateral view, showing the stalk.

of the diverticulum was eventually discovered, lying in close proximity to the descending aorta. The fundus was grasped with a pair of sponge forceps and the diverticulum freed of its surrounding adhesions. When thus displayed the diverticulum was about 2½ in. long, rather like the thumb of a glove (*Fig.* 355). The thin stalk shown on the radiograms was not seen. The neck of the diverticulum was next crushed and ligatured, and eventually buried with a purse-string suture, and finally oversewn with a series of sero-serous sutures. The abdomen was then closed without drainage.

SUBSEQUENT PROGRESS.—The patient made a good recovery and had a comparatively uneventful convalescence. Since operation, twelve weeks ago, he has been quite free from

his previous indigestion and has gained 11 lb. in weight. He volunteers the fact that he has never felt so well in his life.

PATHOLOGICAL REPORT (Dr. John Taylor).—A section of the diverticulum shows that all the coats of the stomach are present.

A further X-ray examination shows that the stalk of the diverticulum is still present in the wall of the stomach (*Figs.* 356, 357).

FIG. 355.—Appearance of diverticulum at operation.

FIG. 356.—After operation, showing diverticular stalk.

A B

FIG. 357.—Aimed exposures showing: A, Mucosal folds passing into stalk; B, Stalk distended with opaque meal.

COMMENTARY

Two types of diverticula of the stomach are described—(1) congenital, and (2) acquired. True congenital diverticula are composed of all the coats of the stomach wall—mucous, submucous, muscular, and serous. Very few diverticula have been described as present in the new-born infant or in children, either at post-mortem or radiologically. It is therefore probable that they are extremely

rare. Diverticula in which the muscular coat is absent are classified as acquired. These represent the great majority and are found in early and later adult life. Most of them are regarded as hernial protrusions of the mucous and submucous layers at a congenitally weakened site in the muscular coat, covered by the serous layer. A few are obviously traumatic in origin, such as the post-operative diverticulum, but these are uncommon.

As regards the acquired diverticula, the prevailing view is that the most important predisposing factor is a congenital weakness or defect in the muscular coat. Diverticula are found in those situations where the muscle fibres are more widely spaced—in the region of the cardia and at the pylorus. Of other factors, that of pulsion is the most important. A herniation of the mucous and submucous coats takes place at the congenitally weakened site as the result of recurrent increases in the intragastric pressure. Once the protrusion has taken place it increases in size as the result of the repeated filling and distension which takes place with every meal. The distension leads eventually to a thinning of all the coats of the diverticulum.

The opening of the diverticulum into the stomach varies considerably, for it may be large enough to admit one or two fingers or it may be filiform in size. The size of the opening is therefore of some importance, for the smaller the opening the greater is the liability to retention of food and the development of complications. Further, the opening may be narrowed as the result of inflammatory swelling of the mucous membrane surrounding it. When a retention persists in a diverticulum, decomposition takes place and inflammatory changes supervene. As the inflammation spreads through the walls of the diverticulum, adhesions between it and neighbouring structures result from the peridiverticulitis. In these cases traction will play a part in accentuating the diverticulum.

Gastric diverticula give rise to no characteristic clinical picture, and in the living person the diagnosis can only be made radiologically. From a consideration of the ten cases it will be observed that the radiological examination was undertaken for one of three reasons : (1) Suspected gastric ulcer (4 cases) ; (2) Suspected duodenal ulcer (4 cases) ; (3) Hæmatemesis (2 cases).

In the first group a gastric ulcer as well as a diverticulum was found in 2 cases. In the second, a duodenal ulcer as well as the diverticulum was found in 1 case. In the third group no lesion other than the diverticulum could be detected. Thus in 7 of the 10 cases under consideration, the diverticulum could be regarded as responsible for the patient's symptoms.

Gastric diverticula are single and vary in size from that of a pea to a plum. They are usually to be found in the region of the cardia, most commonly springing from the posterior wall, but they are found in other situations, although less commonly so—near the pylorus (*Fig.* 358) and on the anterior wall (*Figs.* 359, 360).

Akerlund[1] in 1923 described the diverticulum at the cardiac end of the stomach as a circumscribed, rounded, bag-shaped, projection from the lumen of the stomach, showing varying degrees of distension and filling, and with an absence of radiographic evidence of an infiltrative process in its neighbourhood. When distended it has sharply defined margins and according to the degree of distension its outline may be smooth or lobulated. In the erect position it may show a fluid level, the opaque meal collecting in the lower part of the pouch and the upper part containing air. Diverticula in the region of the cardia are protected by

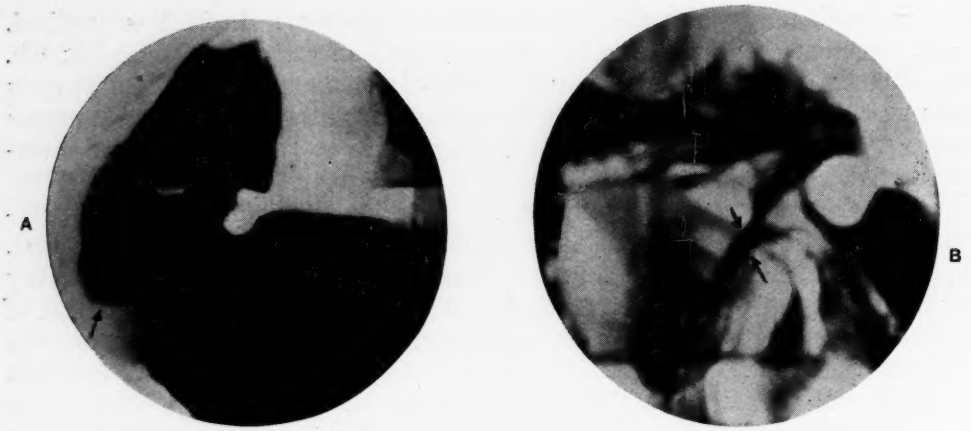

FIG. 358.—Diverticulum of pyloric canal. A, Filled stomach; B, Aimed dosed compression exposure showing mucosal relief of diverticulum, and mucosal folds of stalk.

FIG. 359.—Diverticulum of anterior wall of stomach. Upper arrow indicates ulcer niche.

FIG. 360.—Aimed dosed compression exposures of same case. A, Postero-anterior view; B, Lateral view.

the ribs and are not accessible to palpation. A diverticulum arising from the anterior or posterior wall will be very easily missed if the examination is only carried out in the erect position and with distended filling of the stomach. By using small amounts of opaque meal and by examining the patient in all positions between the Trendelenburg and the erect, the diverticulum can be projected clear of the stomach, and its stalk and point of origin shown. Radiographically diverticula invariably show a stalk, although the stalk may not be apparent at operation or even at post-mortem. The stalk is situated within the wall of the stomach, and on the radiogram it is accentuated by the weight of the barium-filled pouch. At operation and at post-mortem, when the diverticulum is empty and its walls are collapsed, the stalk will not be visible. For the same reason the diverticulum itself may be very difficult to find. Berg[2] and Albrecht[3] have pointed out that in the stomach, as elsewhere in the alimentary tract, longitudinal mucosal folds enter and pass through the narrow stalk. The presence of the longitudinal folds in the stalk is pathognomonic of a diverticulum.

FIG. 361.—Gastric diverticulum, simulating a post-cardiac recess. Lower arrow indicates gastric ulcer.

FIG. 362.—Aimed dosed compression exposure, oblique view, showing stalk of diverticulum and mucosal relief of pouch.

A gastric diverticulum may be mistaken for a large ulcer, particularly a penetrated ulcer. A diverticulum is, however, usually found in a situation where ulcers are rare, and in its immediate neighbourhood there are none of the characteristic changes which are found with an ulcer—convergence of mucosal folds, or concave indentation of the adjacent profile. With an ulcer the mucosal folds converge to the margins of the crater but do not enter into it. Occasionally in the slighter forms of cascade stomach a posterior cardiac recess may simulate a diverticulum. Examination in the oblique and lateral positions and the demonstration of a stalk serves to differentiate a diverticulum from such a recess (*Figs.* 361, 362). Occasionally diverticula in adjacent portions of the alimentary tract—in duodenum and jejunum—may be projected on to the stomach and simulate a gastric diverticulum. They should offer no difficulty in diagnosis, since they can be projected clear of the stomach, and their point of origin shown.

CONCLUSION

It would appear that diverticula of the stomach are not so rare as is generally supposed. They are commonest in the region of the cardia and may be easily overlooked at the radiographic examination. A diverticulum should not be regarded as an incidental finding since it may give rise to severe hæmatemesis or to symptoms of gastric or duodenal ulcer. Mayo[4] has reported a case of a gastric diverticulum which was the site of a carcinoma. The indications for operation would appear to be the persistence of symptoms in spite of medical treatment and the persistence of a retention in the diverticulum after the stomach has emptied.

FIG. 363.—Cardiac diverticulum, showing lobulation.

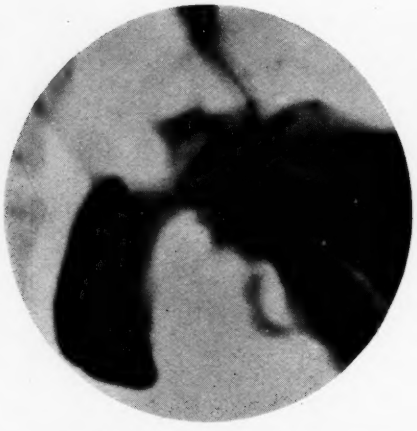

FIG. 364.—Aimed exposure of same case, showing stalk and relation of diverticulum to œsophagus.

Albrecht considers that tenderness over a barium-filled diverticulum indicates the presence of a diverticulitis. *Figs.* 363, 364 show a diverticulum in the cardiac region which was accessible to palpation, was tender on pressure, and in which a considerable retention persisted after the stomach was empty. So far medical treatment has failed to relieve the symptoms.

The actual operation, more especially in the cardiac type, must always be difficult, owing to inaccessibility and the difficulty of palpation of the diverticulum owing to the collapse of its walls. Accurate localization of the diverticulum at the radiographic examination is therefore of the greatest value.

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A METHOD OF TREATING FRACTURES OF THE LOWER LIMB

USE OF A COMBINED COUNTERPOISE AND TRACTION SYSTEM WITH A THOMAS LEG SPLINT AND HINGED KNEE-PIECE ATTACHMENT *

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INTRODUCTION

THE method of treating fractures of the lower limb described by Hamilton Russell[1] combines counterpoise with extension. A travelling pulley to obtain a mechanical advantage is used. The pulley also compensates for the angles assumed. In practice, however, the Hamilton Russell method is unsatisfactory. This is probably because the angle of the knee is not fixed. The weight of the limb is supported by the sling behind the knee. The weight acting at the sling will vary indirectly with the angle between the thigh and the bed. As the patient picks himself up and so alters the angle between his thigh and the bed, the change of strain at the sling is compensated for at the travelling pulley attached to the leg. This produces a further compensatory alteration of the knee flexion. Therefore the moment the patient moves, the non-fixed knee constitutes a third variable factor in the mechanism—that is, the moment the patient moves his thigh from the bed, the mechanism becomes unstable. The patient tries to allow for this instability by altering the angle of his knee voluntarily. This often produces a painful spasm, with possible displacement. Thus only when the angle of knee flexion is constant can the mechanics of the principle of Hamilton Russell be applied.

Another common method is that which applies the Hodgen principle, using a Thomas splint. Here the splint and the limb constitute an entity which is counterpoised by weights—usually hung over the back of the bed. Another distal weight applies 'extension'. There are two great objections to this method. First, the distal or foot end is, as it were, anchored by the weight, applying extension. The ring of the splint and the thigh do not move together unless a considerable upward pull is applied proximally. This often causes undue pressure of the ring behind. Secondly, the direction and the amounts of the counterpoise and extension pulls, vary with the position of the patient in his bed. When the patient gets down to the foot of the bed the counterpoise pull acts to some extent counter to the extension pull.

In treating numerous cases of fractured femurs by the Hodgen principle with a Thomas splint, it was noticed that when the counterpoise weight was applied

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to the long bars of the splint at about the point of balance of the limb, a little proximal to the knee, the patients felt extraordinarily comfortable *until* the extension weight was applied.

The method to be described here was evolved from these observations and was designed to obviate the disadvantages and to use the combined advantages of both the Hamilton Russell and the Hodgen-with-a-Thomas-splint principles.

This method embodies the following features: The limb is fastened to the splintage apparatus. The limb and the apparatus is treated as an entity. This entity is counterpoised by a system of cords and pulleys and a single weight. At the same time and by the same mechanism a pull or 'extension' is applied in the axis of the fractured bone.

The splint with the limb is suspended so that the whole mechanism is virtually a first-class lever. The patient controls the lever by means of that portion proximal to the fulcrum. This proximal arm of the lever is made as short as possible, and its movement is also assisted by a pull transferred over pulleys, so that the patient can control the whole mechanism with the minimum of effort and the minimum excursion of movement.

The counterpoise is of a continuous and compensating nature, so that the patient may move his limb about freely without altering the position in the splint at which the fracture has been set. The patient is thus able to govern his own movements, such as are necessary for nursing, bed-pans, and so on. He can change his position in the bed with only a slight effort and may even sleep on his side without affecting the setting of the fracture.

Results so far have shown that in the fifty-odd cases so treated such a feeling of security and ease is experienced by the patient that he will allow more manipulation and moulding to be done. Consequently with this method the resulting absence of muscle spasm and manipulation possible has given a greater accuracy of anatomical reposition of the fragments. Fine adjustments and settings of angulations, rotations, or other such corrections can be made with every assurance that they will be maintained.

The system is so arranged that the resultant of all the forces and turning moments acting on the limb 'applies extension' in the axis of the fractured bone for whatever alteration in bodily position the patient may undergo. This resultant force can be made greater than that obtained by any direct pull because: it acts indirectly on the 'distal fragment' from many different points of application; the principle of the travelling pulley is used in order to obtain a mechanical advantage; the pull is constant in direction and continuous in application, in spite of the movements of the patient. Finally, no intricate or unusual apparatus is required. Adjustments can be made and kept with mathematical precision.

GENERAL DESCRIPTION OF THE SPLINTAGE APPARATUS

Terms used and Names of Integral Parts.—The terms 'distal' and 'proximal' will be used to designate positions nearer the foot and the head, respectively. Traction distally in the axis of the fractured bone will be called 'extension'. The terms 'upper' and 'lower' will be used to show vertical relations from the floor or bed. The terms 'inner' and 'outer' are used relatively to the sides of the injured limb. The complete splintage, counterpoise, and

traction apparatus will be referred to as the 'apparatus'. The entity of the apparatus with the limb attached will be referred to as the 'mechanism'.

The Three Components of the Apparatus.—The apparatus may be analysed into three components: (1) The splint–knee-piece basis; (2) The suspending cradle; (3) The combined counterpoise and traction arrangement of cords and pulleys.

1. *First Component: the Splint–Knee-piece Basis* (*Fig.* 365).—This consists of the ordinary Thomas traction leg splint, a hinged knee-piece attachment, and a pair of cords. The parts of the Thomas splint to be referred to are: a proximal padded ring, an inner and outer long bar, and a distal cross-bar. The junction of the long bars with the ring will be referred to as the 'ring–long-bar junction'. The knee-piece attachment also has two long bars and a distal cross-bar. The knee-piece may be clamped to the long bars of the splint by means of a screw bolt and hinged plate at the end of each of its long bars.

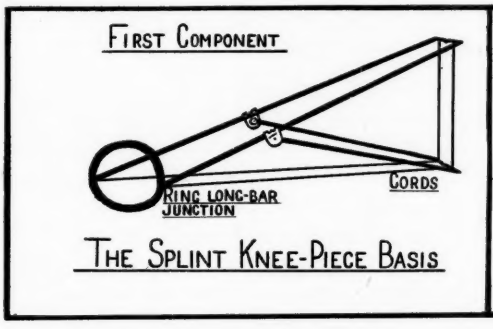

FIG. 365.—Diagram of splint knee-piece basis.

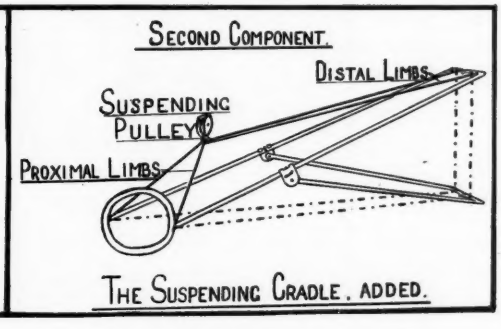

FIG. 366.—Diagram showing the suspending cradle added.

The splint with its knee-piece attached is a familiar method of arranging a fracture so that movement at the knee is possible. In this method this movement is not wanted. The angle which the knee-piece makes with the splint has to be fixed. This is done by the cords. These cords span across, on each side, from the ring–long-bar junctions to the distal cross-bar of the knee-piece and across up to the distal cross-bar of the splint. The distal cross-bar of the knee-piece is thus tied to each end of the splint, and the angle which the knee-piece makes with the splint is thereby fixed.

The splint–knee-piece basis, then, is used as a double inclined plane to which the limb is fixed. The thigh rests in the portion of the splint proximal to the knee-piece and the leg in the knee-piece. When complete the limb lies with the knee flexed at a selected angle fixed by the cords.

2. *Second Component: the Suspending Cradle* (*Fig.* 366).—This consists of a double pulley and two cords. The cords are fastened to the pulley in such a way that, with the pulley as an apex, the cords radiate as four limbs, delineating a pyramidal space. This pulley will be called the 'suspending pulley'. The pair of cords which run proximally from the suspending pulley to the ring–long-bar junctions will be referred to as the 'proximal limbs'. The other pair running from the pulley to the distal cross-bar of the splint will be referred to as the 'distal limbs'.

When suspended by the pulley, the splint–knee-piece basis tends to rotate according to the relative lengths of the limbs. By altering the lengths of these limbs, the suspending pulley at the apex of the cradle is brought farther backwards, distally, proximally, or to one side.

This cradle, then, is the means whereby the fulcrum or point of pivot of the mechanism is regulated. By bringing the suspending pulley proximally, the fulcrum is also brought proximally, and by pulling the pulley nearer to one side the mechanism and with it the ' distal fragment ' of the fractured limb is given a rotation to the opposite side.

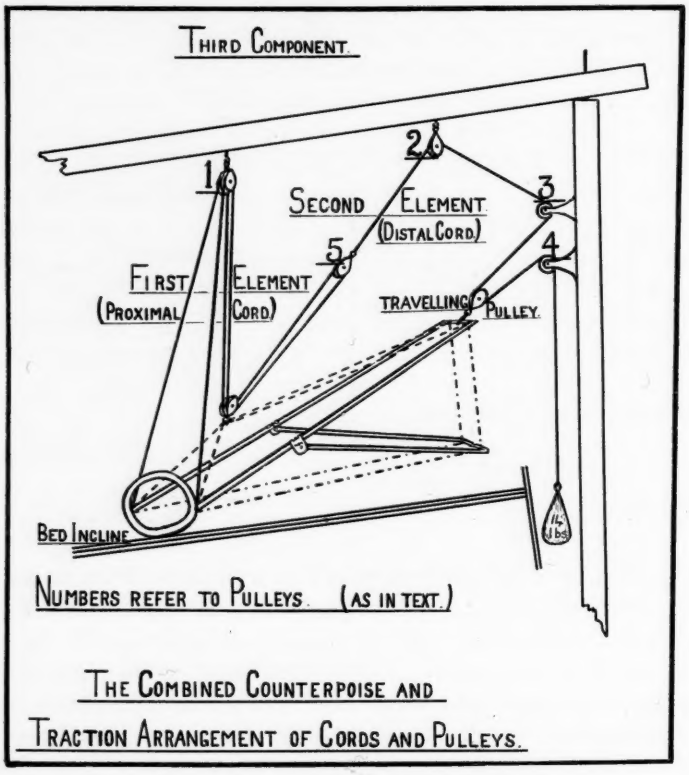

FIG. 367.—Diagram showing the complete apparatus.

3. *Third Component : the Combined Counterpoise and Traction Arrangements of Cords and Pulleys* (*Fig.* 367).—This consists of a ' Balkan frame ', pulleys, and cords.

Frame.—The frame is the usual familiar article of hospital equipment, which provides a horizontal overhead beam and a vertical distal beam.

Pulleys.—To the overhead beam at distances less than the length of the splint are fixed two pulleys : a double one proximally and a single one distally. These will be called ' No. 1 pulley ' and ' No. 2 pulley ', respectively. To the vertical beam are fixed two pulleys, one above the other at a height about 2 ft. above the bed. These will be called ' No. 3 pulley ' and ' No. 4 pulley ' respectively. The positions of these pulleys in actual cases are shown in *Table I.* Their final positions

are fixed when the final adjustments are made. Another single pulley described as the 'travelling pulley' is attached to the distal cross-bar of the splint. An extra pulley is used between the cords to give a compensating sliding attachment. This is termed 'No. 5 pulley'.

Cords.—These comprise two elements, as shown diagrammatically in *Fig.* 367. The first element, or proximal cord, runs from each ring–long-bar junction, up over the double pulley No. 1, back through the double suspending pulley of the cradle, and half-way up to the single pulley No. 2. By the sliding attachment of pulley No. 5, the second element continues the line of the first element up through No. 2 pulley. This second element then continues down around No. 3 pulley, across around the travelling pulley on the splint, back around No. 4 pulley, and so to the weight. The sliding attachment between the two elements is obtained by fastening the second element to the eye of pulley No. 5. This pulley runs in the bight of the first element. The weight used is one of about 10 to 12 lb. and will be referred to as 'the weight'.

The part of this component from No. 3 pulley to the weight is mainly concerned with pulling the mechanism distally. Here the travelling pulley is used to give a true mechanical advantage in producing this pull. The rest of this third component acts mainly as a counterpoise or suspension. It produces an upward pull, but it also produces to some extent a distal pull.

In dealing with a fracture two considerations dominate the procedure. These are the factors of counterpoise and extension, and they must be considered distinctly and individually. Certain cases with deformity and shortening will require the maximum extension, whereas others with good anatomical apposition will not require so much. These latter and also those in the end stages of their treatment may require suspension rather than extension.

Therefore to obtain the *maximum extension* No. 1 pulley must be as far down towards the vertical beam as possible without neutralizing the counterpoise or upward pull unduly. Viewed laterally, the projection down of the line between pulley No. 2 and the suspending pulley should pass proximally to the ring–long-bar junction. The 'afferent and efferent' portions of the cord from either side of the travelling pulley to the Nos. 3 and 4 pulleys should be as nearly parallel as possible. (Because the mechanical advantage gained by means of the travelling pulley is equal to the weight multiplied by a constant, minus an amount for friction. This constant is theoretically equal to two, but it will vary indirectly with the angle of these 'afferent and efferent' portions of the cord to one another.)

With the increase in the extension the foot of the bed must be raised, as the patient's own body weight acts as the counter-extension.

Where conversely the *minimum extension* with suspension is required the cords from the splint and its cradle to the overhead beam may be more vertical. And in the extreme degree of almost pure suspension with no appreciable extension, No. 3 pulley may be dispensed with. The cord then passes from No. 2 pulley straight through the travelling pulley to No. 4 pulley and the weight.

Thus variations to suit the desired requirements of individual cases are possible. Further actions of this component will be considered in the detailed description—that is, with the limb in place and the mechanism controlled by the patient.

DETAILED DESCRIPTION OF THE PROCEDURE OF SETTING UP A FRACTURE

Assembling the Apparatus.—As in the Hodgen principle, the 'distal fragment' has to be fastened to the distal cross-bar of the splint and/or the distal cross-bar of the knee-piece. The hold on the 'distal fragment' may be obtained by adhesive strapping, ice-tong calipers, or other such method which may be in vogue. In this series of cases 35 have been done with adhesive strapping and the other 15 with ice-tong calipers. Using this method, plain strapping applied to the skin produces the minimum of trauma with the maximum of pull, because the pull is obtained indirectly and from numerous sources and different parts of the limb. The strapping may be applied to the whole limb, to the thigh alone, or to the thigh and leg separately. The loop from the thigh turns distal to the knee and above the leg, and is fastened to the cross-bar of the splint. The loop from the leg turns distal to the foot and is fastened to the cross-bar of the knee-piece.

The manœuvres up to this stage may be done with the patient on an emergency splint. After the hold has been secured on the distal fragment the selected Thomas splint with the knee-piece loosely attached is gently worked on to the limb. The knee is flexed as required. Cross-bands of flannel are clipped beneath the limb across the long bars so as to form a trough for the limb to lie in, in the usual manner. Those for the thigh are fastened to the splint and those for the leg round the knee-piece. The ring of the splint is pushed well on to the ischial tuberosity. The knee is slowly flexed to the desired angle, and the knee-piece clamped tight to its proper place on the long bars of the splint.

The cords to complete the splint–knee-piece basis are now fastened in. These are fastened to each ring–long-bar junction and stretched across to the distal cross-bar of the knee-piece, where they are fastened by a clove hitch. These cords are pulled tighter than required and the whole is allowed to rest on the bed while the cords are continued up to be fastened to the distal cross-bar of the splint. These must be pulled tight enough to give a distinct downward bow to the long bars of the splint. Subsequent stretch in the cords will then not jeopardize the rigidity of the splint–knee-piece basis.

The limb now lies in a double inclined plane and has next to be fastened to it. This is done by a cord from 'the hold on the distal fragment' to the distal cross-bar of the splint or the knee-piece, as the case may be. This cord is worked up tight until the ring presses on the ischial tuberosity. Later when the mechanism is working, the distal pull or extension relieves the pressure from the tuberosity.

The suspending cradle is next attached. The suspending pulley is brought over a spot proximal to the point of balance. The proximal limbs will be shorter the more proximal the site of fracture. The reason for this is explained later on. The case examples in the table of measurements illustrate average settings. The limbs are, at this stage, only fastened temporarily, as most of the later adjustment is to be done with them.

The third component is now arranged. This will vary, as stated, to suit the requirements relative to suspension and extension.

The foot of the bed is then put up on blocks. As *Table I* shows, with a weight of 12 lb. and a setting to produce an average amount of extension, a patient of about 150 lb. weight needs the foot of the bed raised at least 10 in.

Nos. 1 and 2 pulleys are fixed to the overhead beam at distances respectively of about 40 and 24 in. proximal to the vertical beam. Nos. 3 and 4 pulleys are set so that the cross-bar of the knee-piece swings well clear of the bed under all conditions. This is of great importance. The whole effect of the counterpoise is neutralized if any part of the mechanism other than the ring rests on the bed.

The travelling pulley is next fastened to the cross-bar of the splint. The cords of the third component are now threaded in. The cord of the first element is fastened to the inner ring–long-bar junction and its other end is threaded in turn through the inner side of pulley No. 1, the inner side of the suspending pulley, through pulley No. 5, and back through the outer side of the suspending pulley and the outer side of pulley No. 1, to finish at the outer ring–long-bar junction. This latter end is finally fastened after all the other adjustments have been made. One end of the second element is now fastened to the eye of No. 5 pulley, and the other end is passed in turn as has been described through the remaining pulleys to the weight.

Adjusting the Apparatus. Consideration of the Mechanics.—The mechanics must be understood in order to make the adjustments. This entails a little digression.

The portions of the limb proximal and distal to the fracture will be referred to as the ' proximal ' and ' distal ' fragments. The ultimate objective is to suspend the mechanism so that it behaves as a first-class lever—that is, there must be a fulcrum or neutral point about which the mechanism rotates, a proximal arm and a distal arm. As the site of fracture and the fulcrum are made to coincide as nearly as possible, reference to the 'proximal arm' and 'distal arm' will imply the portions of the mechanism in relation to the respective proximal and distal fragments.

Considering the suspending or counterpoise element alone : When the proximal arm moves up the distal arm moves down. With the patient lying relaxed in the supine position the weight of the thigh keeps the ring of the splint on to the bed. When the patient raises his buttocks from the bed the mechanism must tip up so that the ring will follow the thigh without lag or undue pressure in any direction. (Discrepancy between the movements of the ring and thigh means, of course, that there is movement between the ends of the fragments.)

The weight of the distal arm imparts a downward rotation. Some pull is thereby given to the cord of the second element. This in turn imparts an upward lift to the suspending pulley. Now this suspending pulley is also a type of travelling pulley. Another mechanical advantage, therefore, increases the pull transferred to the apex of the suspending cradle. Thus the weight of the distal arm, by a mechanical advantage gained at the suspending pulley, helps to produce at the ring–long-bar junction an increased upward pull on the proximal arm. The lighter distal arm can thus be made to counterbalance the heavier proximal one. The excursion of the distal arm will naturally have to be greater than the proximal one. This, then, is a mechanism which the patient can control by a force applied only at the proximal or ring end.

Considering the whole mechanism with the distal traction or extension element added : The distal end is, as it were, anchored by the force applying extension. This force acts through the medium of the travelling pulley. This ' anchoring ' force can be analysed into a horizontal and a vertical element (Nos. 3 and 4 pulleys

are higher than the travelling pulley). The latter vertical element of the force tends to lift the distal up or prevent it from depressing. This tends to disturb the movements of the system we have just considered. It tends to convert the lever into a beam supported at two places. The mechanism, however, will again behave as a lever when only this vertical element is neutralized. This is done by moving the suspending pulley proximally. In other words, a tendency must be established for the moments which depress the distal arm to preponderate. This tendency neutralizes the vertical element of the 'anchoring' force. Thus by merely shortening the proximal limbs there is no longer the same 'anchoring' effect. The movements of the mechanism are again those of a counterbalanced lever. The patient is able to tip the mechanism in the same way as before the force applying extension through the travelling pulley was added. The distal arm moves up and down to correspond with the movements in the opposite direction of the proximal arm, the difference now being that the whole fulcrum appears to travel up farther and its site is less definite. An important point is that the movement of the distal arm will be easier, the greater the distance between the Nos. 3 and 4 pulleys.

Actual Adjustments.—The adjustments are made with the co-operation of the patient. It is essential that all his movements should be painless and free. Settings for two positions are effected. These positions are :—

No. 1 *Position.*—Supine with buttocks and ring of the splint on the bed.

No. 2 *Position.*—With buttocks raised, as for the insertion of a bed-pan.

The patient moves himself into these positions with the aid of a 'monkey chain' fixed over the head of the bed. The mechanism is viewed from the side. As the proximal end moves up, the distal end should depress. The point at which the mechanism appears to rotate should be about the site of fracture. If this point is too far distal, the ring of the splint will lag behind the thigh as the No. 2 position is assumed. The correction will be to shorten the proximal limbs of the cradle—that is, to bring the suspending pulley proximally. The opposite fault, causing the ring to press in posteriorly, is unusual. The suspending pulley is then too proximal.

With fractures above the upper third of the femur the suspending pulley may be as far proximal as possible and yet the ring may lag behind the thigh. The further correction then will be to bring No. 1 pulley proximally.

With fractures proximal to the ring, such as the central dislocation of the hip, movement at the fracture cannot be obviated. The best procedure then is to minimize the effort required to produce movement, while the hip-joint may be kept fixed. The neutral point in these cases is best placed just proximal to the knee.

When a fracture is below the knee the distal fragment is fastened to the knee-piece. The travelling pulley is also fastened to the distal cross-bar of the knee-piece, which comes to lie more horizontal. Flexion at the hip-joint is increased. The proximal limbs of the cradle will be longer than the distal limbs (*see Table I*).

Extra flannel bands may now be placed around the limb so as to correct wrong angulations or rotations. These with the cross-bands forming the trough are now finally adjusted and fastened with large safety-pins. Attention is paid to the 'master band' which preserves the anterior bow of the femur.

The limb and the splint may be next wrapped round with a bandage. This bandage is taken up to and woven across the front part of the ring of the splint. Any further alteration to the third component may now be done. Any gross alteration of this component may entail a corresponding alteration to the second component. Finally, the cord comprising the first element of the third component is adjusted and fastened so that the No. 5 pulley is mid-way between the No. 2 and the suspending pulleys.

The patient now performs all his movements, pulls himself to the top of the bed, and by externally rotating at his hip he may roll over to the unaffected side. The bedclothes are tucked around and over the mechanism so that only the cords to the Balkan frame emerge from the covers. This depresses the mechanism very little. The distal end of the knee-piece may have attached the usual foot-piece which holds the foot at a right angle. The distal end is inspected to see that the cross-bar of the knee-piece does not touch the bed. Should this bar even come near to the bed in the No. 2 position, the correction will be to lower the foot of the bed, raise one or both the Nos. 3 and 4 pulleys, or to increase the weight. Should the excursion of the distal end not be sufficient, the correction will be to separate the Nos. 3 and 4 pulleys, having made certain that the suspending pulley is as far proximal as is feasible.

After a day or so the pull distally on the mechanism relieves the tuberosity of the ischium of the pressure of the ring. Should this become exaggerated and the ring pull away by more than two finger-breadths, the cord holding the distal fragment to the cross-bar must be shortened.

The attachments of the knee-piece to the splint take the stress and strain of the whole mechanism. They are crucial points. It is essential that they grip absolutely tightly to their positions on the splint. Later with shrinkage of tissues, pulling out of shortening, correction of deformity and so on, further adjustments such as tightening or replacing bands and readjustments to the strapping may become necessary.

Much later, when union is established, suspension rather than extension may be required. This is provided for by the alteration already described, up to the extent of eliminating No. 3 pulley.

FOUR TYPICAL CASES ILLUSTRATING FOUR DIFFERENT TYPES OF FRACTURE

For comparative purposes as well as for conciseness the weights, dimensions, angles, and other data concerning these four cases have been condensed in the form of a table (*Table I*).

Case 1.—**Fracture of the middle third of the femur.**

A. T., aged 29. Weight estimated at about 140 lb. Admitted on May 29, 1934, with a fracture of the left femur and other wounds. Adhesive strapping was applied and the limb fixed to the splint–knee-piece basis, while the patient was recovering from his shock. Later he was transported on this basis to the theatre and back. The rest of the counterpoise traction mechanism was completed when the man was returned to bed. He was not X-rayed prior to being put up.

From the beginning he was able to move himself around without any discomfort. As there was no shortening to be overcome there were no further adjustments to be made to

the cord from the strapping to the cross-bar of the splint. On the sixth day the antero-posterior and lateral X-rays were taken (*Figs.* 368, 389). These are as shown. Next day the photographs of the man in the No. 1 and No. 2 positions were taken (*Figs.* 370, 371). After the seventh week the patient left hospital in a weight-bearing caliper, and by the sixteenth week he was able to take weight directly on the limb, and he reported back in the twentieth week walking normally without a limp.

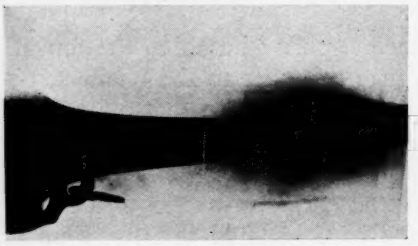

FIG. 368.—*Case* 1. Antero-posterior X-ray photograph after treatment.

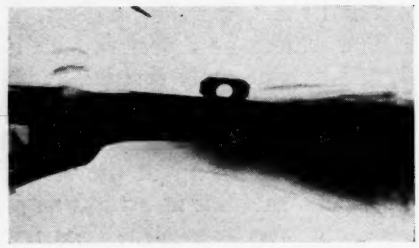

FIG. 369.—*Case* 1. Lateral X-ray photograph after treatment.

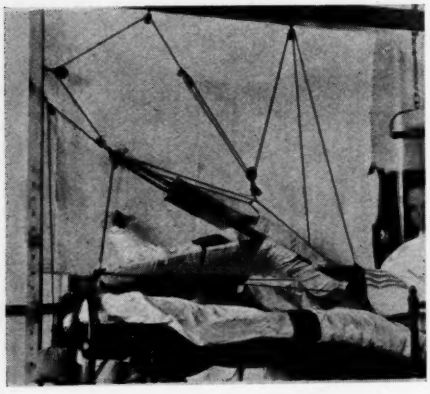

FIG. 370.—*Case* 1. Photograph showing patient in the No. 1 position.

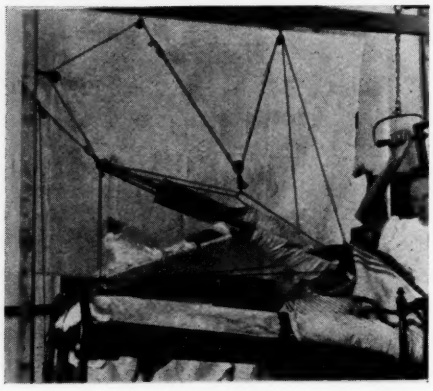

FIG. 371.—*Case* 1. Photograph showing patient in the No. 2 position.

Case 2.—**Fracture of the lower third of the femur.**

W. I., aged 55. Weight 165 lb. before accident. Admitted on June 23, 1934, with a fracture of the right femur. There were numerous abrasions on the leg, and the knee was

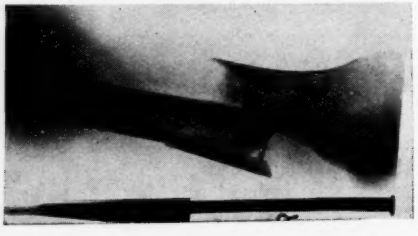

FIG. 372.—*Case* 2. X-ray photograph, almost antero-posterior, before treatment.

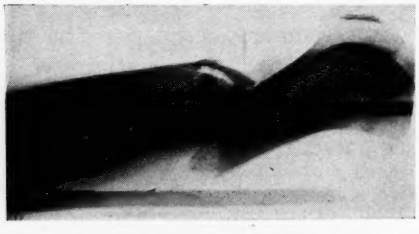

FIG. 373.—*Case* 2. X-ray photograph, lateral, before treatment.

distended with blood. He was treated by a method of fixed traction, pending the attention of his Benefit Society doctor, for two weeks. X-ray photographs were taken on June 25. These show the case 'before treatment' (*Figs.* 372, 373).

On July 4 the case was officially handed over as a hospital patient. The old strapping was removed. There were numerous sores on the leg. There was about 2½ in. of shortening. Ice-tong calipers were inserted into the femoral condyles and the limb was put up in the counterpoise traction mechanism. Daily readjustments were made to keep pace with the correction of the shortening. On July 11 the condition was stationary, and X-rays and

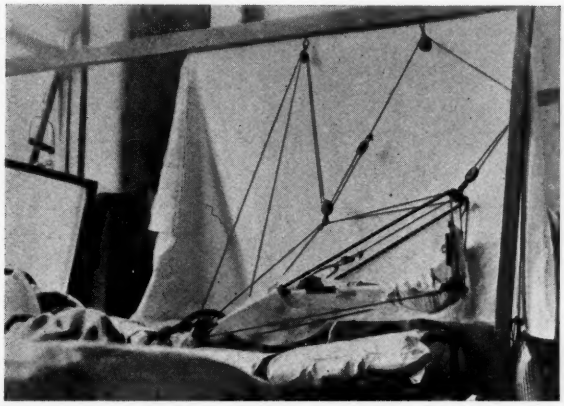

FIG. 374.—*Case* 2. During treatment. Note the use of the ice-tong calipers.

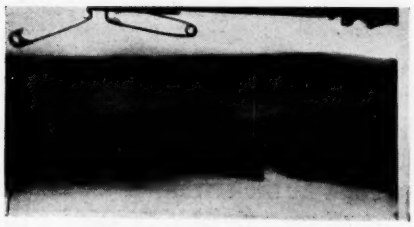

FIG. 375.—*Case* 2. X-ray photograph, antero-posterior, after treatment.

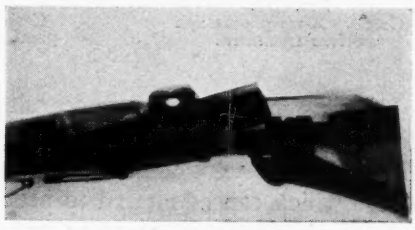

FIG. 376.—*Case* 2. X-ray photograph, lateral, after treatment.

photographs were then taken (*Figs.* 374–376). By Aug. 14 the splint–knee-piece basis had been altered so that the patient's knee had been moved to an almost completely extended position. On Nov. 27 the man was walking normally and could flex his knee through 170°–100°.

Case 3. **Fracture of the upper third of the femur.**

W. W., aged 30. Weight about 160 lb. Attended on April 5, 1932, at the Nursing Home, Springs, Transvaal. The fracture was then three weeks old. As much as 30 lb. weight had been applied by direct extension at some time. There was still 2 in. of shortening. On April 5 the limb was put up in the counterpoise traction system. Strapping extension was used. On April 10 the cord to the distal cross-bar of the splint had been shortened by 1¾ in. The patient meanwhile permitted fairly extensive daily manipulations. By Oct. 1 the man was back at his work as a miner. He then had a good alinement with about ½ in. of shortening.

Case 4. **Fracture of the middle third of the leg.**

J. F., aged 19. Weight about 120 lb. Admitted on Oct. 20, 1934, with a compound fracture of the leg. He was cleaned up and put up in a split plaster soon after admission. On Oct. 23 the leg was transferred to a Thomas splint as the wound required frequent dressings. On Oct. 26 the fracture was in very poor position, as can be seen from *Fig.* 377. On Oct. 27 the limb was put up in the counterpoise traction system. Ice-tong calipers were

inserted into the malleoli. The travelling pulley was fastened to the distal cross-bar of the knee-piece. The patient was X-rayed again on Oct. 29. This X-ray shows that there was nearly ½ in. of separation between the fragments (*Figs.* 378, 379). This is particularly interesting as the weight used was only 5 lb. No. 2 pulley was accordingly shifted proximally 12 in. and the cord from the distal cross-bar of the knee-piece to the caliper lengthened by ½ an in. The boy left hospital in a plaster on Nov. 17.

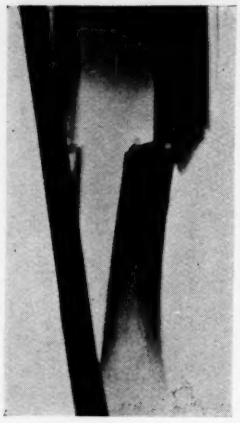

FIG. 377.
Case 4. X-ray photograph before treatment.

FIG. 378.
Case 4. Lateral X-ray photograph after treatment.

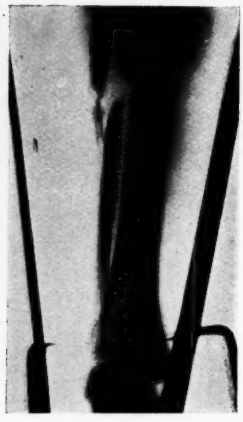

FIG. 379.
Case 4. Antero-posterior X-ray photograph after treatment.

In *Table I* the data concerning the splint–knee-piece basis, the suspending cradle, and the non-variable factors of the third component are grouped together. These do not vary with the change in position of the patient. These are the factors which can be altered to produce an alteration in the setting of the fracture. The remaining factors grouped under the heading of the ' variable factors ' change with the positions assumed by the patient, but no alteration of the setting of the fracture can occur. In the third case the angles and, by means of spring balances, the tensions in the various segments of cord have been measured and plotted. From the readings parallelograms of forces were constructed to represent the magnitude and direction of the forces acting, when the patient was in the No. 1 and the No. 2 positions. Without knowing the weight of the limb it was possible to calculate the turning moments of the mechanism. The final resultant of the forces could be shown to be exerting a pull distally in the axis of the fractured bone for both the positions.

In practice, however, this mathematical result is obtained by merely altering the adjustments provided until the patient says that movements are painless and that he feels quite safe and rigid and is being pulled strongly down towards the vertical beam of the Balkan frame. One patient said, " It feels as though I am being hung up by the leg ". He felt most of the pull at the posterior part of the calf. The extension on the mechanism may by pressing on the calf hold the leg forward. A flannel band to counteract this is placed over the *front* of the distal third of the leg. This is an example of the production of an indirect pull on the femur exerted through the ligaments of the knee-joint. This pull will vary directly with the angle of the knee-piece to the splint. (Compare the angles in *Cases* 1 and 2.)

Table I.—DATA CONCERNING FOUR CASES OF FRACTURE OF THE LOWER LIMB

Case Numbers	1		2		3		4	
1st Component—								
Length of splint	39·3 in.		40·0 in.		39·6 in.		39·3 in.	
Length of knee-piece	24·5 in.		24·0 in.		24·0 in.		24·0 in.	
Angle of knee-piece to splint	42·4 °		28·0 °		39·0 °		36·0 °	
Angle of lower cords to splint	26·8 °		17·2 °		22·5 °		21·8 °	
Angle of vertical cords to splint	67·8 °		77·3 °		76·0 °		76·5 °	
2nd Component—								
Proximal Cords—								
Average of lengths	18·5 in.		18·0 in.		9·9 in.		32·6 in.	
Inclination to splint	23·2 °		20·6 °		31·2 °		14·7 °	
Distal Cords—								
Average of lengths	23·4 in.		24·0 in.		31·4 in.		11·2 in.	
Inclination to splint	17·3 °		15·2 °		9·4 °		46·3 °	
3rd Component. Non-variable Factors—								
Weights used	10 lb.		12 lb.		12 lb.		5 lb.	
Inclinations of beds (to horizontal)	6·0 °		7·3 °		7·3 °		5·0 °	
Distance proximal from vertical beam of—								
No. 1 pulley	38·3 in.		40·5 in.		38·5 in.		39·5 in.	
No. 2 pulley	15·5 in.		25·0 in.		24·0 in.		22·0 in.	
Height up from distal surface of bed of—								
No. 3 pulley	33·5 in.		31·0 in.		33·6 in.		27·4 in.	
No. 4 pulley	27·0 in.		27·7 in.		26·3 in.		18·0 in.	
3rd Component. Variable Factors—								
Position numbers	1	2	1	2	1	2	1	2
Height buttocks lifted from bed	0 in.	5 in.	0 in.	4 in.	0 in.	3 in.	0 in.	5 in.
Inclinations of splint (to horizontal)	36·3 °	23·6 °	30·5 °	22·0 °	30·1 °	25·3 °	36·2 °	25·9 °
LENGTHS AND INCLINATIONS OF LINES BETWEEN—								
Ring–long-bar junction and No. 1 pulley—								
Length	38·3 in.	33·8 in.	36·3 in.	33·2 in.	39·4 in.	37·1 in.	41·2 in.	37·4 in.
Inclination	88·5 °	82·4 °	77·1 °	76·0 °	76·8 °	75·5 °	76·5 °	71·6 °
No. 1 pulley and the suspending pulley								
Length	22·2 in.	20·2 in.	20·0 in.	19·0 in.	28·6 in.	26·6 in.	16·9 in.	17·8 in.
Inclination	67·3 °	65·6 °	80·2 °	73·7 °	97·8 °	96·7 °	51·3 °	45·6 °
Suspending pulley and No. 2 pulley								
Length	25·8 in.	24·2 in.	25·0 in.	23·2 in.	35·0 in.	33·0 in.	15·2 in.	13·6 in.
Inclination	56·8 °	53·6 °	61·5 °	64·7 °	58·5 °	57·8 °	62·9 °	69·8 °
No. 3 and the travelling pulley								
Length	11·8 in.	14·0 in.	16·6 in.	17·1 in.	18·2 in.	17·8 in.	20·1 in.	22·7 in.
Inclination	58·9 °	70·2 °	44·0 °	53·0 °	51·3 °	55·0 °	60·8 °	60·6 °
Travelling pulley and No. 4 pulley								
Length	7·2 in.	8·5 in.	14·5 in.	14·6 in.	13·3 in.	12·5 in.	12·8 in.	15·2 in.
Inclination	29·3 °	51·0 °	34·7 °	45·1 °	31·3 °	34·5 °	39·5 °	43·0 °

The technique used with the calipers and the adhesive strapping may be epitomized thus :—

Calipers.—The application of this implement is treated as a surgical operation in the theatre. Wounds are first attended to, dressed, and bandaged. The surgeon then cleans himself afresh, and likewise the sites for the insertion of the calipers, and a fresh set of instruments is used. The caliper after insertion is well packed round with sterile dressings and a plentiful supply of sterile cotton-wool. A sterile bandage is woven between the tongs of the caliper until the whole is enclosed in dressings, with only the end for the attachment of the cord uncovered. The subsequent removal of any other dressings must not disturb in any way the dressings around the calipers.

Adhesive Strapping.—The salient features here are: Collars of padding are stitched into place over the bony prominences, e.g., the femoral condyles or malleoli. Main straps are placed in the usual manner along the sides of the limb, forming a loop distally. These straps may be taken well above the site of fracture. Their ends are ribboned for about one foot or so. Short narrow strips are then applied so as to cover in spica fashion the main ones, the limb finally being almost encased in a mould of adhesive strapping. The ribboned ends of the main straps are turned back and incorporated with the short spica strips. The whole is next covered with a well-fitting puttee of flannel, stitched into place. The usual rectangular 'spacer' piece of wood is fitted into the loop of the main straps. The position of this spacer is determined after a few 'trial-and-error' pulls after the whole has been completed. The cord which fastens the distal fragment to the distal cross-bar of the splint is fixed to this rectangular block of wood and threaded through the loop of the main straps.

CONCLUSION

A method of treating fractures of the lower limb is described. It is particularly intended for fractures of the femur from the upper third distally and for fractures of the upper half of the leg.

The main features are the great amount of ease, comfort, and relaxation experienced by the patient, and the fine degrees of adaptability, regulation, and control which can be effected and maintained.

The method has been used for both hospital and country patients who had to be treated in their homes. The upkeep and after-care is minimal. In the 50 cases so far treated by this method, anatomical replacement has been good in all but 2 cases. Fractures with 2 in. of shortening have been pulled out in a few days. The average time taken completely to assemble and adjust the mechanism has been just under an hour. Subsequent adjustments were all performed in a few minutes. The house surgeons who have been shown how to use the method have had no difficulties, and their results have been good.

Finally, no intricate or unusual apparatus is required. Operative measures and anæsthetics have been seldom called for. Plain strapping applied to the skin has given the maximum of pull with a minimum of trauma, because the pull is obtained indirectly and from numerous sources and from different parts of the limb.

I would like to thank Professor Brebner for his kindly criticism and for the opportunities he has given me to try this method on the patients admitted under his care; Mr. F. P. Fouche, who taught me the technique and niceties of the Hodgen principle applied to the Thomas splint; Professor Gordon Grant for his valuable criticism and advice concerning the presentation of this paper; Messrs. Moller and Mackenzie, Honorary Surgeons to the Germiston Hospital, on whose patients the method was evolved. I am greatly indebted to Professor Dart and Drs. Galloway and Gillman, of the Anatomy Department, for their enthusiasm, editorial advice, and photographic activities, without which this paper would not have been written. I am also indebted to Mr. Kay and Mr. Mason and Miss Tomkins for their help with the photographs and X-rays.

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RENAL RICKETS AND DWARFISM: A PITUITARY DISEASE

By BRUCE CHOWN

CHILDREN'S HOSPITAL OF WINNIPEG, AND GRACE HOSPITAL, IN ASSOCIATION WITH THE MEDICAL COLLEGE OF THE UNIVERSITY OF MANITOBA

In the past two years I have seen two babies, sisters, having from birth deformities like those of severe rickets. A few days after birth they began to have curious spells of sham joy. Both had a hypercalcæmia but normal or slightly reduced phosphatæmia without discernible progressive decalcification of the skeleton. One died at three months and the other at six. Both had early pathological changes in the kidneys, which would have led to chronic nephritis. At first I thought that these were cases of congenital osteitis fibrosa cystica, but now believe that the two children were suffering from a form of the disease variously called 'renal rickets', 'dwarfism', or 'infantilism'. If this is true, then the cases add weight to the opinion that these diseases are due to endocrines running amok, the kidneys being but innocent bystanders.

CASE REPORTS

Family History.—The parents, Mr. and Mrs. L., are first cousins of English-Welsh extraction. There are no dwarfs or giants in the family so far as is known. Mr. L. has been married twice. His first wife, pregnant but once, bore him a son, and died four years later of some acute febrile illness. The son has a boy of four years. These two, the son and grandson, are apparently normal. Mr. L. was 33 years old, Mrs. L. 20 years, when they were married in 1916. The record of Mrs. L.'s pregnancies is as follows :—

1	2	3	4	5	6	7	8	9
E+O	●	●	●	♂	♂	♀	GWEN	JEAN

E+O = Ectopic and removal of blood cyst of ovary

● = Miscarriage. 5 and 6 were stillborn. 8 and 9 are the cases here reported

In the first two years after her marriage her teeth "went chalky" and were pulled. She weighed 115 lb. in 1916, 183 lb. in 1927, and now weighs 159 lb. She is an active, bright, intelligent woman of 38, normal on physical examination; blood Wassermann repeatedly negative; basal metabolic rate −12; calcium and phosphorus balance normal; X-ray pictures of skull, pelvis, arms, and legs normal. Her menses began when she was 15 and have been regular. She has always suffered from severe premenstrual occipital headaches. When she is pregnant she feels particularly well; never suffers from nausea, vomiting, salivation, sweating, or excessive dryness of the skin; her hair grows long and thick, only to fall again after childbirth. Mr. L. is a rather small 52-year-old normal man of 125 lb.; blood Wassermann negative. Their 6-year-old girl is normal.

Case 1.—Gwen (C.H. No. 32–3039). In this pregnancy Mrs. L. was admitted to hospital at the seventh month and again at the eighth with false labour pains. The baby, however, was born at term after a four-hour labour; weight 7 lb. Deformities, although undoubtedly present, were not noted at birth. At 10 days she began to have convulsions, for which she was admitted to the Children's Hospital at 3 weeks. She weighed 6½ lb.; temperature 101° to 102°, then normal; pulse 114 to 140. There were present a maculo-papular eruption over her legs and feet, slight internal strabismus, pliable cranial bones such as one often finds in a newborn premature infant, beading of the ribs, enlargement of wrists, anterior angulation of both tibiæ (*Fig.* 380). An area of skin 3 × 4 mm. over either point of angulation was hairless, smooth, and adherent to the tibia; in comparable areas over each fibula was a small pit overlying an exostosis, while over the posterior aspect of the right ulna was a small hairless area neither adherent nor depressed. Owing to a short metacarpal bone the right middle finger was the same length as the fore and ring fingers. The left second toe crossed over the third. The lymph nodes and liver were not enlarged; the spleen and kidneys not palpable. The knee-jerks were hyperactive; Chvostek's and Trousseau's signs absent. There was a moderate polymorphonuclear leucocytosis. The spinal fluid was said to contain 59 cells per c.mm., 70 per cent polynuclears, but I think this doubtful. The Wassermann reaction was negative in both blood and spinal fluid. X-ray examination showed widespread bony lesions (*Fig.* 381), which were then interpreted as probably syphilitic. After four days' observation the child was sent home on bottle feedings and antisyphilitic treatment. She had only one minor convulsion in hospital.

FIG. 380.—*Case* 1. Showing enlarged wrist and angulated tibia.

FIG. 381.—*Case* 1. X-ray at 3 weeks of femur, radius, and ulna, showing marked rachitoid cupping.

At home she took her artificial feedings eagerly, and, though she vomited, her mother thought she was doing well. Then one night five weeks after discharge she uttered loud cries. These were repeated the following night, while on the next day "she cried out like an idiot and threw her arms and legs around or would contract her whole body." (This type of spell was observed in detail in her sister and will be described in a separate paper.) She was re-admitted to hospital the next day. She now weighed 6 lb.; temperature 101° to 104°, then down to normal with an occasional rise to 101°; head 13¾ in., chest 12¾ in., abdomen 11 in., against a normal 14½ in. for each of these. The baby's general condition was unchanged from her previous admission. Once more the spinal fluid was reported to contain an increased number of cells. In the blood there was a marked polymorphonuclear leucocytosis—36,700, with a slight anæmia—3,800,000. The urine twice contained a trace of albumin and once a few pus cells. The blood calcium was 15·5 and 15·8 mgrm. per 100 c.c., the phosphorus 5·4. She took her milk badly, vomited much, gained about 2 oz. a week. At times her colour was grey; she cried out, moved her head from side to side, or waved her hands as though distressed. After two and a half weeks in hospital her temperature rose suddenly to 104°; she was cyanosed, almost continually on the move, cried out. In two days her temperature was down to 101° but there was no other clinical improvement. She vomited blood. Three days later she was dead.

Case 2.—Jean (C.H. No. 34–8391). During this pregnancy Mrs. L. took calcium and viosterol. She was admitted to Grace Hospital at the eighth month with false labour pains, but again carried to term, being delivered of a 7 lb. 6 oz. girl after a two-hour labour. The baby, like her sister, began on the 10th day having convulsive seizures, which appeared to be controlled by calcium. She was sent home and given calcium and viosterol, but these were soon stopped, since it seemed probable that they might lead to calcification of soft tissues. Through the generous co-operation of Drs. F. A. Benner and H. Medovy, I saw the baby when she was 5 weeks old. Her blood calcium was 17 mgrm., and her phosphorus 4·8 mgrm. per 100 c.c., her mother's 10·5 and 2·1. She continued well, though without any gain in weight, till she was 7 weeks old. Seizures then recurring, she was admitted to Grace Hospital under the care of Drs. Benner and Medovy, and a week later was transferred to the Children's Hospital for investigation.

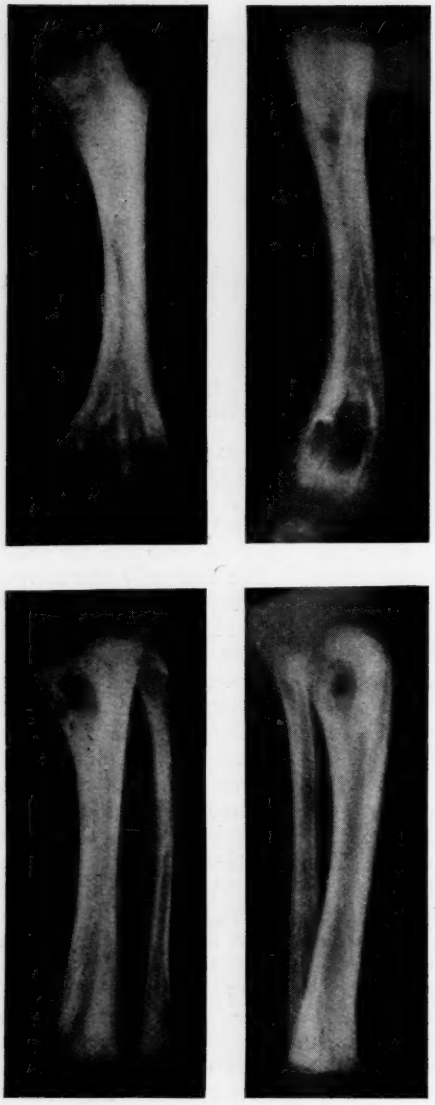

FIG. 382.—*Case* 2. Antero-posterior and lateral X-ray views of femur, tibia, and fibula. While the anterior view of the femur suggests a lesion somewhat like that in *Case* 1, the remaining ones are suggestive of cyst formation, but the rarefactions represent osteoid. This picture was constant from first observation at 5 weeks to death at 6 months.

She now weighed 7 lb.; temperature 100° F; head 14¼ in., chest 12½ in., abdomen 13½ in., length 21½ in. She was well proportioned; eyes round; skin soft and dry; cranial bones widely separated (?); marked beading of ribs; liver edge 1 in. below the costal margin; both kidneys palpable at the level of the navel, and hard; spleen not palpable; wrists and ankles not enlarged. X-ray examination showed widespread bone lesions (*Fig.* 382). Her nervous manifestations will be considered separately. She took her feedings very slowly; was extremely constipated; lost 4 oz. the first week, and looked dehydrated. In view of this and the fact that dehydration sometimes takes place in hyperparathyroidism, she was at Dr. Medovy's suggestion, given extra water by stomach tube. The result was astonishing. She gained 10 oz. in four days and her fever fell to 100°. But, though water was continued, the fever again rose, and up to the time of her death she had recurrent bouts. A general view of her subsequent course may be gathered from *Fig.* 383. Certain special features will be dealt with separately.

Autopsies.—Both bodies were examined after death. Although carefully searched for I found only two minute parathyroid glands in each case. There certainly was no parathyroid tumour or hyperplasia in either. Serial sections of the pituitary in the first case were not obtained. The available sections are histologically normal. In the second, serial sections proved the presence of hemi-agenesis of the partes posterior et intermedia (*Fig.* 383). Histologically the gland was otherwise normal. The thyroids, too, were histologically normal, but the second had small bilateral accessory lobes

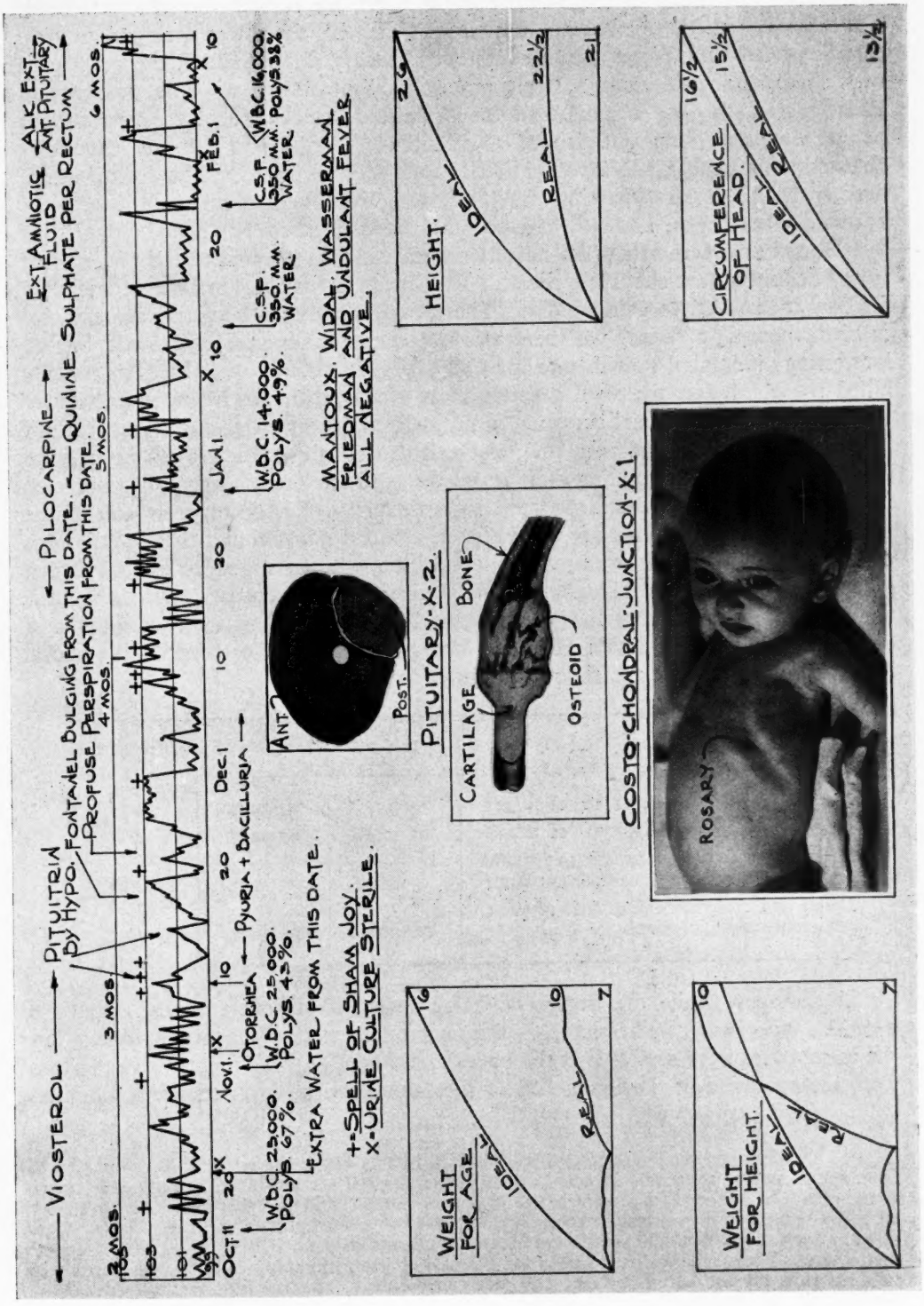

FIG. 383.—*Case* 2. Showing the bone lesion, the pituitary lesion, and the course of the disease.

sticking forward from the edge of the lateral lobes like blinders on a horse's head. The thymus in the first case was small; in the second it weighed 7 grm. Microscopically the cortex and medulla were clearly differentiated, the corpuscles very small and pale with scarcely any eosinophil substance. The ovaries and adrenals in both were normal. In the brain and spinal cord of the first patient no pathological lesion was found. The brain of the second was not externally diseased, but histological investigation is not yet completed. The heart, lungs, liver, pancreas, gastro-intestinal tract, ureters, and bladders in each case were normal. The bones, kidneys, and pituitary were diseased.*

Bones and Growth.—We have not sufficient data relevant to the first patient to say definitely that she was a dwarf. All we can say is that, in spite of adequate feedings, she failed to gain weight. The second patient, although of average size at birth, became a dwarf, yet there was not complete absence of growth, but an interesting differential growth-rate (*see Fig.* 383). It would appear that this actually killed the child, for, the skull growing more slowly than the brain, there was an ever increasing intracranial pressure, with final respiratory paralysis. This in itself is curious. In an infant with progressive hydrocephalus skull growth keeps pace with the size of the expanding brain, even though bone growth may not quite keep up with it. But here there appeared to be inability of bone to grow and of the bones of the cranial vault to separate, for at autopsy it was found that the margins of the bones of the vault were really in apposition, a broad edging of osteoid having given, during life, the semblance of wide separation of the sutures. In six months she grew approximately 1¼ in. (32 mm.), or ⅜ in. (19 mm.) in the four months of observation in the Children's Hospital, but growth was of cartilage and osteoid, not bone, as shown by the following figures :—

	Sept. 14, 1934	Jan. 10, 1935	Growth
Length of shadow—diaphysis of femur	74 mm.	74 mm.	0
Length of shadow—diaphysis of tibia ..	67 mm.	67 mm.	0
Distal end of femoral diaphysis to proximal margin of epiphysial ossification centre	3 mm.	7 mm.	4 mm.
Distal end of femoral diaphysis to proximal end of tibial diaphysis	28 mm.	35 mm.	7 mm.

At autopsy, while the degree of lesion varied in different bones, there was a similar type in all. The shafts of the long bones were of normal hardness, but the metaphyses were so soft that the bones easily bent at these points. The lesions were most marked at the lower end of the femur and at costo-chondral junctions

* The horizontal section of the pituitary shown in *Fig.* 383 is the result of a reconstruction from serial sagittal sections. It can be seen that a sagittal section taken through the centre of the gland, or to the right of centre, would appear normal, and also that absence of posterior lobe in a section to the left might carelessly be passed as due to post-mortem technique. The bulging back of the left half of the anterior lobe made the gland outline so slightly irregular that, though I was looking particularly for a lesion of the pituitary, I did not realize one was present until all the serial sections had been examined. The sections were cut by Miss Service, of the Department of Anatomy of the University, through the kind co-operation of Professor Inkster.

(*see Fig.* 383). On section there was a greater or less accumulation of osteoid in the metaphyses and at the epiphysial line, irregularity of the epiphysial cartilage, and in some cases a transverse vascular zone between cartilage and bone. There were no cysts.

Histologically there was: (1) Delayed senescence of cartilage, so that (*a*) long tongues of uncalcified cartilage extended into the osteoid, and (*b*) small islands, both calcified and uncalcified, persisted within trabeculæ at some distance within the shaft; (2) Failure of the zone of preliminary calcification to form; (3) An enormous overproduction of osteoid; (4) Some focal increase, in the osteoid area, of osteoclasts without fibrosis. The condition is not identical with avitaminotic rickets since there is no great increase in the depth of proliferating cartilage, while there is a tendency to the formation of islands and peninsulas of cartilage. This difference has been noted before in renal rickets, the best microphotograph that I know of being that of Vogt's[1] patient 1.

Mineral Metabolism.—The following observations were made:—

Name	Age	Date	Ca	P	Remarks
Gwen ..	2 months	16/7/32	15·5		
		19/7/32	15·8	5·4	Died 4/8/32
Mrs. L.	36 years	18/1/33	9·9	2·7	
	38 years	14/9/34	10·5	2·1	Not nursing
Jean ..	5 weeks	14/9/34	17·0	4·8	
		10/10/34	14·9		After convulsion
		13/10/34	18·0	3·9	
		19/10/34	15·4	3·0	After convulsion
	3 months	29/10/34	15·3	3·4	
		5/11/34	15·0	3·3	
	4 months	26/11/34	14·3	3·5	Urea nitrogen 12·4
	5 months	3/1/35	17·5	4·0	
		14/1/35	16·7		C.S.F. Ca. 7·6
	6 months	29/1/35	18·4		C.S.F. Ca. 5·5

Daily Balance

Mrs. L., Oct. 1934				Jean, Nov. 1934		
Intake	Output	Balance		Intake	Output	Balance
2·09	1·64	+ 0·45	Calcium	0·72	0·40	+ 0·32
	S. 1·29 U. 0·35				S. 0·35 U. 0·05	
3·29	3·29	+ 0·005	Phosphorus	0·59	0·36	+ 0·23
	S. 1·08 U. 2·21				S. 0·18 U. 0·18	

S = Stool U = Urine

Urinary Tract.—A trace of albumin was present in each specimen of Gwen's urine and a few leucocytes in one. In all of Jean's there was a trace of albumin, a specific gravity of 1005 to 1010. Four cultures at monthly intervals were sterile, though there were usually from one to eight cells per high-power field in the uncentrifuged urine. In her third month *B. coli* was grown from the urine, and for a short time in her fourth month, when clinically she was better than usual, the urine contained *B. fæcalis alkaligenes* and 20 to 30 cells per high-power field. At this time the cells were mainly pus cells, but at other times there were also large and small round cells and giant cells 30 μ in diameter with a single round nucleus. At times coarsely granular casts varying in size and number were found in fresh specimens, but not, as a rule, in routine samples collected one night and examined next morning. On the following grounds these were considered to be a calcium salt in an undetermined matrix: (1) The upper part of the centrifuged urine contained less calcium than the lower, e.g., 12·03 as compared with 16·37 mgrm. per 100 c.c.; (2) They stained black with silver nitrate in the light; (3) They stained red with alizarin; (4) They disappeared when suspended in urine of P_H 4·8; (5) They persisted when suspended in urine of P_H 8·0; (6) When they were acidified with weak acetic acid under the microscope the granules disappeared, leaving transparent rods like slightly crumpled cellophane.

Neither baby was thirsty or voided large quantities of urine. During her fourth month Jean's blood-urea nitrogen was 12·4 mgrm. per 100 c.c.

In both babies the kidneys reached to the umbilical plane and felt hard and lobulated. No special note was made of the state of the ureters and bladder in Gwen's case, and post mortem the kidneys appeared normal in the gross. Jean's bladder was not dilated according to X-ray examination, while both it and the ureters were normal post mortem. Her kidneys were pale, hard on section, and weighed together 69 grm., against the normal of 51 grm. for her age or 40 grm. for her weight.

Microscopically* (*Fig.* 384) the same types of lesions were found in both cases,

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* Since the lymphatics are not recognized as playing a part in the physiology of kidney function, my interpretation of the abnormal deposits in these kidneys as being calcium in lymphatics is open to question. There can be no question of their presence. Part stains black when treated with silver nitrate in the light, part red with alizarin, part has the microscopic configuration of calcium carbonate, and part takes a bluish-purple stain with hæmatoxylin. All this suggests a complicated substance of protein-calcium-phosphate-carbonate nature or a mixture of substances. Further they do not lie in the tubules but in juxtaposition to them, often surrounding a tubule like a collar of beads. In shape they vary from round to long oval. About some a membrane with flattened nuclei can be seen. These circumscribed calcium-salt deposits must derive their elements eventually from the blood-stream. They may either be directly deposited from the blood into these peritubular spaces, or else filtered from blood-stream to glomerulus to tubule, then passed back through tubular epithelium into these same spaces. There seems no reason why the first should take place. The second process is much more consonant with modern physiological theory even though it diverges from that theory in introducing the lymphatics, as intermediaries for some substances at least, between tubule and vein. In the present cases the excessive amount of calcium in the blood leads to an abnormal amount in the glomerular fluid. Part of the excess calcium in the primary urinary filtrate is passed back into the lymphatics. Calcium differs from most re-absorbed substances in readily forming an insoluble salt. This it has done here, the precipitate remaining as evidence of what has been going on. What part the lymphatics play in the normal physiology of the kidney is not known. In preliminary work, in association with Miss Margaret Lee and Mr. John Teal, lesions similar to those here described have been readily produced in young rats by injections of parathormone. A further study of renal calcinosclerosis is in progress and will be reported later.

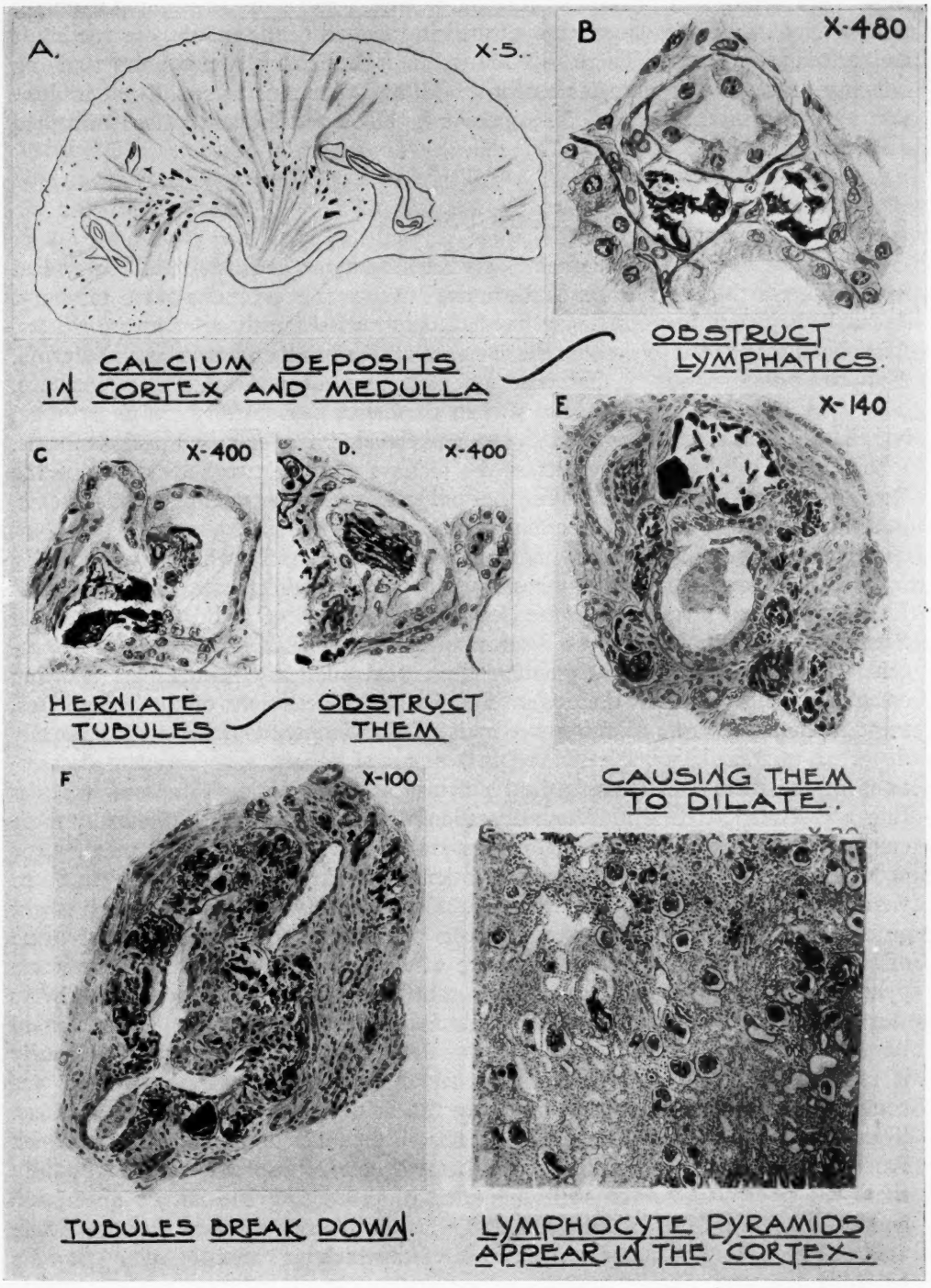

FIG. 384.—Early stages in the evolution of the kidney lesion. (Owing to the reduction of the illustration, the magnifications shown should be approximately halved.)

but they were more advanced in the second. In lymphatics accompanying tubules there were deposits of calcium salts alone or with a hæmatoxylin-staining material. As the deposits became larger they pushed in the wall of the tubule, obstructing it and breaking it down. There was low-grade inflammation, the tubules draining inflammatory cells, albumin, and débris. Minute pyramids of round-cell infiltration appeared in the cortex. The glomeruli, blood-vessels, and pelves remained essentially normal.

DISCUSSION

We may assume the two babies suffered from the same disease. They had from before birth malformation of the partes intermedia et posterior of the pituitary. At birth they were of normal size but had deformities like those of severe rickets. After birth they failed to grow at the average rate, or in other words became dwarfs. They had many nervous manifestations referable to diencephalo-pituitary imbalance. They had hypercalcæmia, calcium casts in the urine, calcium deposits in the renal lymphatics, focal necroses in the kidneys, and the elder one so-called pyelonephritis. I believe the calcareous fragments in the kidneys of these two babies may be the Rosetta Stone of renal rickets, leading not only to a true interpretation of this disease, but also to an explanation of certain other renal ruins.

The concept of renal rickets is of very recent origin, for while Lucas[2] in 1883 recorded the association of albuminuria with rickets in adolescents, it was not until Fletcher[3] showed a case before the Section for Diseases of Children of the Royal Society of Medicine in 1911 that interest was aroused. Later an occasional adolescent with acutely developing genu valgum died suddenly in uræmia following osteotomy. Post mortem there were found rickets-like lesions of the metaphyses, most marked as a rule at the lower ends of the femora, together with strangely distorted, gnarled kidneys. Barber,[4] in a series of papers, gave excellent descriptions of the disease and emphasized polyuria as a presenting symptom, whereas Ellis and Evans[5] stressed moderate dilatation of the urinary tract. Gradually there emerged a picture of rickety deformities occurring in infancy or at puberty, or occasionally after an acute illness, the patients later dying of renal failure. Some were dwarfs and some sexually infantile. Often there was a history, which might go back for years, of great thirst and of the passage of copious quantities of urine of low specific gravity, containing a trace of albumin. In some cases there was a pyuria. There might be hypertension, sometimes with arteriosclerosis. Always there occurred the gnarled kidneys and the leather-tipped bones. There could be no doubt the two were in some way causally related, and, since rickets ordinarily is not recognized as producing any kidney disease, the reverse relationship was considered probably the true one: the chronic nephritis caused the rickets. Shipley[6] and his co-workers suggested a chemical explanation of the disease which Parsons[7] developed: the kidneys could not excrete phosphate, so the gut did; there the phosphate united with the calcium, preventing the latter's absorption and so causing a "low calcium rickets." Parsons further showed that there were at least two types of the disease, or two diseases which appeared the same but were probably different. Teall[8] confirmed this from an X-ray standpoint. The dwarfing was looked upon as due also to the chronic nephritis, being likened to the dwarfing of congenital heart disease.

There was, as Hunter[9] and others have pointed out, much against the explanation of the disease being symptomatic of renal failure. It is impossible to extend the chemical theory to the fœtus so as to account for congenital deformities. Ordinary chronic nephritis is not, either in adult or child, associated with rickets even when there is phosphate retention; where rickets and chronic nephritis are associated the latter is always of a peculiar type. Nephritis has never been proved to produce dwarfing, much less infantilism. The urinary tract dilatation remains an unexplained observation.

Albright, Baird, Cope, and Bloomberg[10] have suggested that the disease in some cases may be really an infantile form of hyperparathyroidism. They cite Parsons's[7] case who had a hypercalcæmia, and their case, K.S. This girl of 13 had had a diagnosis of renal rickets made previously, but on the basis of hypercalcæmia an exploration was carried out, a single parathyroid tumour found and removed. The calcium now fell, the phosphorus rose, and the girl developed chronic tetany and cataracts. Functionally she had only one parathyroid. Radiologically she showed a bone lesion very similar to that in patients with renal rickets, whereas, as the authors point out, other children with hyperparathyroidism have not shown this X-ray picture.

Hutinel[11] reported a girl who at 3 years suffered for fourteen days from headache and vomiting; at 8½ years she was treated for genu valgum; polyuria and polydipsia developed; at 11 years she had albuminuria and œdema. When seen at 13 she had rachitic bone changes, the urine was abundant and contained albumin, and renal function was impaired. He believed the dystrophy was caused by alteration of the glands of internal secretion and particularly the hypophysis (Mitchell's[12] summary).

I believe that Hutinel's interpretation of his case was the correct one and, as Harris[13] suggested, renal rickets is primarily a pituitary malfunction, which may, like any pituitary disease, have certain polyglandular variants. Such an explanation fits well with observed facts, which are difficult to explain on the basis of primary chronic nephritis: the polyuria, the dwarfing, the infantilism, the urinary tract dilatation. The first three are well recognized as pituitary-diencephalic in origin; a word as to the fourth. The bladder and ureters receive both sympathetic and parasympathetic fibres. Without entering into the controversy as to the rôles either section of the autonomic plays in urinary tract contraction and relaxation, I think one may state the following as facts. Dilatation of the bladder sometimes occurs following injury to the spinal cord (sacral autonomic fibres) and may be relieved by resection of the presacral nerve[14] (thoraco-lumbar outflow). Relaxation or contraction of the kidney pelvis, ureters, and bladder can be brought about at will by drugs acting on the autonomic nervous system.[15] Sympathetic and parasympathetic centres exist in the diencephalon.[16] "The diencephalo-hypophyseal can only be properly interpreted when looked upon as a whole." (Cushing). Therefore a worn or ill-made part in this mechanism might produce not only diabetes insipidus (diencephalon), dwarfing (growth-promoting factor of the anterior pituitary), infantilism (sex-stimulating factor of the anterior pituitary), but also urinary tract dilatation (diencephalon). This may sound very theoretical, but in the second case here reported there was such an ill-made part associated with dwarfing and congenital rickets. Langmead and Orr's[17] patient with renal infantilism and late rickets had a malformed pituitary and hyperplasia of the parathyroids.

The nephritis can be explained as the result of calcuria and phosphaturia. In the presence of an increased excretion of these minerals part is passed out in the urine, but part is re-absorbed through the tubular epithelium to the peritubular lymphatics. Here, whether from concentration, change in reaction, or what not, calcium is precipitated as the insoluble calcium phosphate and carbonate. The deposits obstruct first the lymphatics and then, by pressure, the tubules. Some tubules and their glomeruli atrophy from complete obstruction. Some dilate from partial obstruction. In some areas comparable to those shown in *Fig.* 384 F the epithelium grows back again, but there remain sacculations or so-called cysts.[18] The ill-drained and badly damaged kidney becomes a *locus minoris resistentiæ*. Wave after wave of acute interstitial nephritis (pyelitis[19]) comes and goes, leaving destruction in its wake, but also dissolving some of the tell-tale stony fragments. At last the kidney 'fails'. Phosphates are no longer excreted. Calcium and phosphorus are diverted more and more to the gut. Depletion begins. Both physiological and pathological sources are drawn upon. The deposits in the kidneys disappear, and when the child dies of uræmia there remain only the dwarfed and misshapen corpses of the kidneys. It may be that I am wrong in trying to wash the mineral deposits out of these failing kidneys. They quite possibly persist, but, occurring in association with fibrosis, have been looked upon as an end-result of the sclerosing process and so not mentioned. Thus Mitchell[12] only says of his case, R.P., "There was fibrous change in the interstitial tissue of the kidney", yet the microphotograph appears to show calcium in this interstitial tissue (? lymphatics).

The drama of hyperparathyroidism has so fixed its characters in the medical mind that every calcuria cloaks a parathyroid villain, and "*Cherchez la hypercalcæmie!*" is the watchword of every detecting laboratory. A false syllogism has been set up. "All hypercalcæmics have calcuria. Patients with renal rickets have no hypercalcæmia. Therefore patients with renal rickets have no calcuria." The major premiss is true but not exclusive. Calcuria does occur in the presence of hypercalcæmia, but it may also occur in its absence. Thus in Cushing's[16] Case 15 of pituitary basophilism with a blood calcium of 9·8, Aub, who studied the patient metabolically, found "the calcium output [in the urine] was far in excess of normal". A normal calcium figure for the blood does not prove that the kidneys are not being attacked under this flag of peace. It is to be recalled, too, that in renal failure the blood-calcium level falls and the phosphorus rises, so that a hypercalcæmia may be camouflaged in this way. Further, in renal (pituitary) rickets it is not necessary to think of the process as continuous. The calcuria may only appear at the springing-up periods, and indeed a case such as Langmead and Orr's[17] can only be explained on the assumption of a noxious agent acting intermittently. Their patient was first seen at 14 with retarded growth and development and chronic nephritis but no rickets. Six years later he rapidly developed genu valgum and died of renal failure. The chemical theorist would explain this as a chronic nephritis which first retarded body growth, and only later, when renal failure had reached the stage of phosphate retention, caused demineralization and rickets. It would be hard to gainsay such a possibility, were it not that this very case showed at autopsy a malformation of the pituitary and enlargement of the parathyroids. The endocrinophil may here visualize the pituitary spring flood of infancy carrying down silt to clog the kidneys; then a long drought, with stunted growth, the dry period extending far past its wonted time; at last the torrential

pituitary rains of adolescence, washing deep gulleys in the hills and sweeping the débris down to complete the destruction of the kidneys.

But there are a few cases on record of renal rickets which had a hypercalcæmia at one time or another. Karelitz and Kolomoyeff[20] observed a child from the age of 4½ to 6 years. When she was first seen her blood calcium was 10·6, her phosphorus 8·0, and her urea-nitrogen 143. She was given viosterol. The calcium rose to 11·0, phosphorus dropped to 4·2, and urea-nitrogen to 73. According to X-ray examination her bone lesions improved. Viosterol was stopped. Calcium continued to rise to 12·4, phosphorus held at 4·4, and urea-nitrogen fell to 54. Viosterol was started again, to be shortly replaced by cod-liver oil. In six months the calcium was 9·4, phosphorus 7·8, and urea-nitrogen 171. The disease progressed to death six months later. Before death calcification of blood-vessels and lymph nodes was made out, which had not been observed five months before, whereas the bone lesions had advanced with remarkable rapidity. In Duken's[21] patient in uræmia, when viosterol was given, the bones healed, calcium rose from 10 to 16, phosphorus fell from 8 to 6, and non-protein nitrogen from 107 to 30. In Parsons's[7] patient, H.T., who had a blood calcium of from 10 to 14·6, phosphorus of 4·1 to 10·07, and a urea of 112·9 to 136·5, the above relationship did not hold, the calcium, phosphorus, and urea rising and falling together. This patient, Parsons thought, was made worse by ultra-violet radiation. Incidentally Teall reported the X-ray picture of the skull in this case as " identical with that of Paget's disease". Lightwood's[22] delightfully reported " Case of Dwarfism and Calcinosis " had a blood calcium of only 11·0 but a phosphorus of 6·68 and urea of 169 a few days before death, yet the fibrotic kidneys contained deposits of calcium in tubules and in glomerular remnants. The parathyroids were reported as definitely not enlarged, but the pituitary was not mentioned. I think the 11·0 mgrm. of calcium per 100 c.c. in the presence of renal failure must here be interpreted as a masked hypercalcæmia, and that this case was a variant of pituitary rickets. In Karelitz and Kolomoyeff's patient there appeared to be first an improvement and then a rapid progress in the disease. Their case, it seems to me, can be interpreted somewhat as follows: Calcinosis of the kidney, the result of pituitary rickets, had gone on to beginning failure. Viosterol caused at first withdrawal of minerals from soft tissues, including kidneys, to bone (X-ray evidence). The kidneys, relieved of the mineral obstruction, improved in function, with consequent rise in calcium and drop in phosphorus and urea. The continued use of vitamin D produced the reverse effect, the bones lost calcium, the soft tissues, including the kidneys, took it up, renal function fell rapidly, and the child died in uræmia.*

Such a result has been reported in experimental hypervitaminosis D.[23] There is an analogous parallel for this reversal of action in work emanating from Collip's

* The orthodox teaching at the moment is that vitamin D controls absorption or excretion of calcium through the intestinal wall. In massive doses, however, it produces decalcification of bone, and in continued small doses increased calcification, i.e., a systemic effect. The above case suggests a sensitivity to vitamin D, just as Parsons thought his patient was made worse by ultra-violet light, so that an ordinarily physiological dose produced the effects of massive doses. Wilder, Higgins, and Sheard[25] have suggested that the parathyroids determine sensitivity to vitamin D, sensitivity to the vitamin increasing in parallel with activity of the gland. Ham and Lewis[26] have produced in rats a lesion indistinguishable from rickets by the exhibition of ergosterol.

laboratory, Selye[24] having shown that whereas parathormone first caused decalcification of bone, continuation of the extract produced hypercalcification. Smyth and Goldman[27] recorded a remarkable case of a boy who developed rickets and enormous deposits of calcium phosphate following a streptococcal septicæmia. He died of renal failure. At autopsy the parathyroids were enlarged, while during life his blood calcium had run around 12 mgrm. and his phosphorus from 12 to 16 mgrm. The pituitary was not remarkable in the gross. This case, like Hutinel's,[11] may, I think, best be looked upon as a counterpart of Simmonds's disease, an injury to or functional depression of the pituitary causing in one case an upset in mineral metabolism, in the other in fat metabolism. Smyth and Goldman were of the opinion that the enlargement of the parathyroids in their patient was a secondary phenomenon.

This brings us back to the possible relationship of the parathyroids. In the case of baby Jean the small calcium output and good retention were surprising. There was also a good phosphorus retention, but the normal partition of stool 2 to urine 1 has been changed to a 1 to 1 relationship. This latter taken with the blood chemical findings is suggestive of hyperparathyroidism. The pituitary probably has a parathyrotropic function.[28, 29] Collip[30] has brought evidence to show that any particular hormonal level of activity depends upon a balance between a hormone and its anti-hormone. In the present case the chemical findings of hyperparathyroidism could be brought into agreement with the histological evidence of normal parathyroids by assuming the absence of the parathyroid anti-hormone. One would have to go farther and assume that since there was mal-development of the pituitary the latter was the governor of anti-parathyroid hormonal activity. I must admit, however, that I do not find the position a happy one. On the other hand, it is to be noted that histologically there is no evidence of osteoclastic release of calcium, the osteoclasts occurring among osteoid trabeculæ. The hypercalcæmia can be ascribed simply to normal absorption with failure of deposition. The parathyroids might then become drowsy owing to the sedative action of the calcium, so that one could conceivably have a hypoparathyroid state in the presence of a hypercalcæmia.

Up to this point, then, evidence and argument have been brought to show that the minor symptoms of renal rickets may best be accounted for as due to pituitary mal-function, and the nephritis to a calcuria or phosphaturia, which in turn is the result of faulty bone metabolism. The argument must finally turn on the relationship of the pituitary to the bone lesion, and here proof fails. Bone metabolism is affected by mineral intake, vitamins, inflammation, trauma, the parathyroids, the gonads, the thyroid, and the pituitary. The lesion in question simulates or actually is rickets, which in turn can be produced experimentally by avitaminosis D, by hypervitaminosis D,[26] by thyroidectomy,[31] by parathyroidectomy.[32] Rickets, ordinary avitaminotic rickets, is in some way related to growth, for if growth ceases the rickets will heal,[13] and is also related to sex, for it is more common and more severe in males.[33] Renal rickets, too, manifests itself at what Harris[13] calls " the springing-up periods ", the times of rapid (pituitary) growth. But, curiously, growth does not take place; there is preparation for growth but failure of the bones to add to their length. There is tubulation without elongation. This suggests that there are normally two factors active in bone growth and that here one has failed. As fast as bone grows at the epiphysis it is remodelled at the

metaphysis. At puberty, in health, these two processes speed up *pari passu*. Suppose them governed by the pituitary through two of its subsidiaries, then if it were at fault it might speed up the process of metaphysial resorption (perhaps via parathyroids) without building new bone (perhaps via thyroid). Osteoid would be thrown out to buttress the weakened bone. The minerals made available by resorption would be excreted by the kidney. Renal calcinosis would develop. The picture is complete. This speculation is best closed by again quoting Harvey Cushing: "Of all subjects that engage the attention of the profession at the present day, that of endocrinology particularly lends itself to the temptation of impressionistic speculation." Amen.

The argument in proof of the thesis that a lesion of the pituitary-diencephalic mechanism is the primary cause of the symptom complex called renal rickets is, then, as follows: (1) Malformation of the pituitary has been found in these cases; (2) The associated symptoms of dwarfing, polyuria, infantilism, and urinary tract dilatation can be caused by such a lesion; (3) The nephritis is not primary but is secondary to an abnormal mineral metabolism, itself the result of faulty bone growth; (4) The faulty bone-growth therefore not being due to the nephritis, and the remaining symptoms being due to pituitary-diencephalic disease, it is to be presumed that the bone disease is due to the same cause. Should this argument prove correct, it offers some hope for these previously hopeless cases, for substitution therapy could in theory produce normal bone growth in the springing-up periods and so save the kidneys from being stoned to death.

In the preparation of this paper I have had assistance from many. The first case was brought to my attention by the late Dr. Andrew Sykes, and it is to him too that I owe the early obstetrical history of the mother. Recognition of the second case depended upon the clinical acumen of my associate Dr. Harry Medovy, while it was through his generosity, and that of Dr. Frank Benner, that the patient was turned over to me for investigation. To Professor A. T. Cameron I am indebted for overseeing the metabolic studies carried out by Miss Margaret Lee, B.Sc.; for estimating the basal metabolism of Mrs. L.; for doing a Friedman test; and for his critical interest. I am grateful to Miss Margaret Richardson for the many X-ray studies she carried out for me, to Mrs. Helen Ross for the photographs, to Mr. John Teal for the innumerable microscopic sections he prepared, to Dr. Anna Wilson for much help and friendly criticism, and last but not least to Miss Fannie McLeod, R.N., Nurse in charge of Infants' Ward, Children's Hospital, and to her pupils, for the unremitting care they bestowed on baby Jean, and for the meticulous accuracy of their observations and records.

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ISOLATED DISLOCATION OF THE BASE OF THE FIFTH METACARPAL

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CARPO-METACARPAL dislocations of the thumb are relatively frequent, and in combination with a Bennett fracture through the base of the metacarpal constitute a common injury. Isolated dislocations of the second, third, and fourth metacarpal bones have been reported on numerous occasions, but a careful search of the literature reveals only one previously reported case of isolated dislocation of the fifth metacarpal on the carpus. McWhorter,[1] who reported this case in 1918, had been unable to find any record of a previous case.

In addition to isolated dislocations of individual metacarpals, multiple dislocations occur, and simultaneous dislocation of all five metacarpal bones has been described (Burk[2]). In a review of the literature, Burk quotes twelve cases of dislocation of the base of the fifth metacarpal in association with other dislocations. In seven cases it was dislocated dorsally, completely in each case, and in five cases anteriorly, two being incomplete.

Speed[3] states that no case of isolated dislocation of the fifth metacarpal has been recorded, and other text-books on fractures omit reference to the condition or state that it is unknown. Stimson[4] does not mention the condition.

The writers have been able to collect radiographs of four cases and the clinical details are available in all but one.

Case 1. (C. T. H.'s case).—This patient, a man aged 53, in getting off a bus fell heavily on his left side with his left hand between his body and the roadway. He noticed that his little finger looked to be "out of place", and went to his doctor, who "put it straight". Five days after the injury the whole hand was considerably swollen, the fingers stiff, and bruising was present over the inner metacarpal region. A fracture of one of the inner metacarpals was suspected.

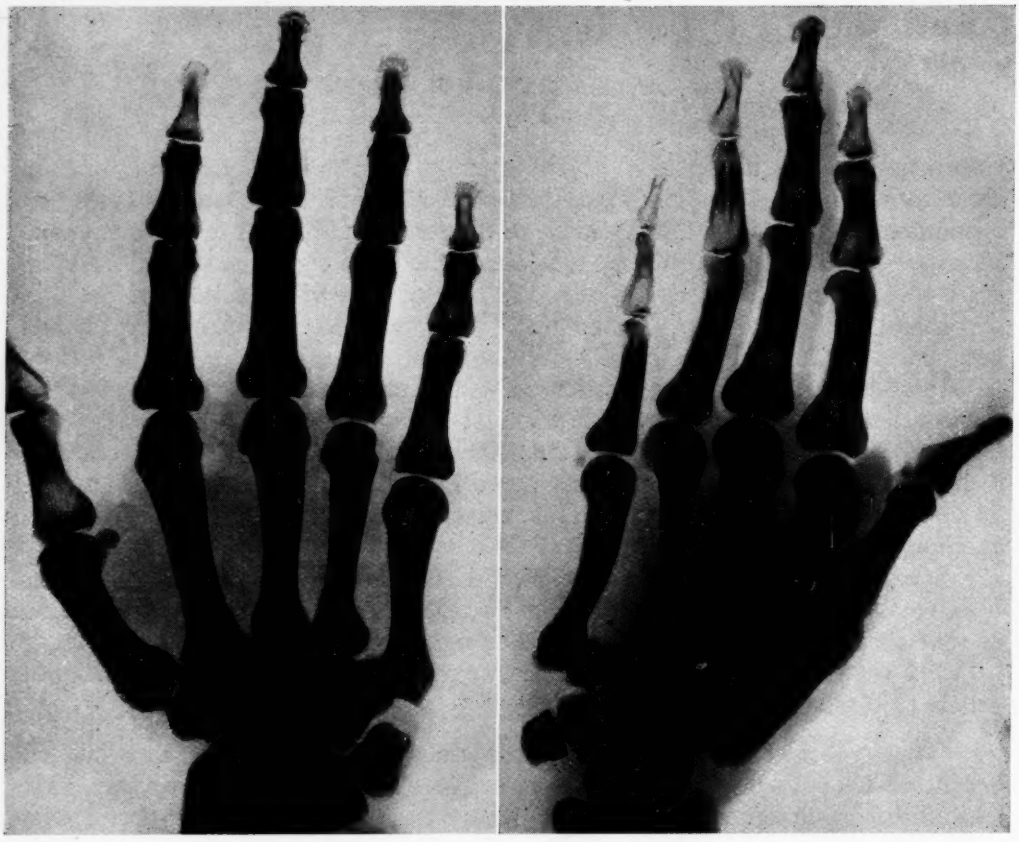
The radiographs (*Fig.* 385) show a complete dislocation of the base of the fifth metacarpal, the latter being displaced inwards and forwards with consequent shortening of the finger.

Attempts at reduction failed entirely, and radiographs taken later on showed the condition to be unchanged. Five months after the accident there was almost complete functional recovery, his doctor estimating it as a 95 per cent good result.

Case 2.—The details and radiographs of this case have been obtained by one of us (C. T. H.) through the kindness of Dr. Sankey, of Oxford. The patient, an adult male, injured his hand in March, 1931, and was found to have dislocated his fifth metacarpal. The radiograph (*Fig.* 386) shows an inward dislocation of

the fifth metacarpal with shortening of the finger, the base of the metacarpal lying immediately to the ulnar side of the unciform.

Attempts to reduce the dislocation failed, but when he was re-examined in September, 1933, he was found to have excellent function. The grip on the inner side of the hand was slightly impaired but there were no painful symptoms. The patient was following his normal employment as a bus cleaner and had not lost a day's work in consequence of the injury.



A B

FIG. 385.—*Case* 1. Dislocation of fifth metacarpal bone: A, Hand, palm down; B, Semi-lateral view.

Case 3. (N. R.'s case).—The patient, a woman aged 31, slipped and fell on the left hand on Jan. 21, 1935. The diagnosis was not made on clinical examination, but when the radiographs (*Fig.* 387) had been taken the patient was re-examined more carefully and was found to have definite shortening of the finger with a painful and unduly prominent base of the fifth metacarpal.

Reduction was easily accomplished by traction on the finger combined with local pressure over the metacarpal and no anæsthetic was necessary since the finger was relatively painless. On releasing the traction the dislocation immediately recurred. The finger was splinted on a wire frame splint with traction on the finger obtained by skin extension, and in this position the dislocation remained reduced

completely. Traction was maintained for three weeks and then discontinued under the impression that the reduction was stable. However, when the patient was re-examined on June 28, redisplacement had occurred, but not to the extent of the original displacement. The functional result was excellent, the only abnormality being a tendency for the finger to drift into a position of ulnar abduction when the hand was relaxed. Full active adduction of the finger was possible.

Case 4.—In this case, from the late Dr. Robert Knox's collection, no clinical details are available beyond the fact that the patient was an adult male. The illustration (*Fig.* 388) is one of a pair of stereoscopic radiographs which show that

FIG. 386.—*Case* 2. Dislocation of fifth metacarpal bone: hand palm down.

the proximal end of the bone was lying in front of the bases of the third and fourth metacarpals.

The first three cases are almost identical, the base of the metacarpal being displaced inwards and forwards. Reduction of the displacement is easily accomplished, but unless continuous traction is exerted the dislocation recurs. This is accounted for by the fact that the articulation relies for its stability entirely on its ligamentous connections. In order to maintain a stable reduction it would probably be necessary to continue immobilization with traction for four to six weeks.

The fourth case is matched by McWhorter's case, the only other previously reported. His patient, a man aged 23, sustained a dislocation of the fifth metacarpal

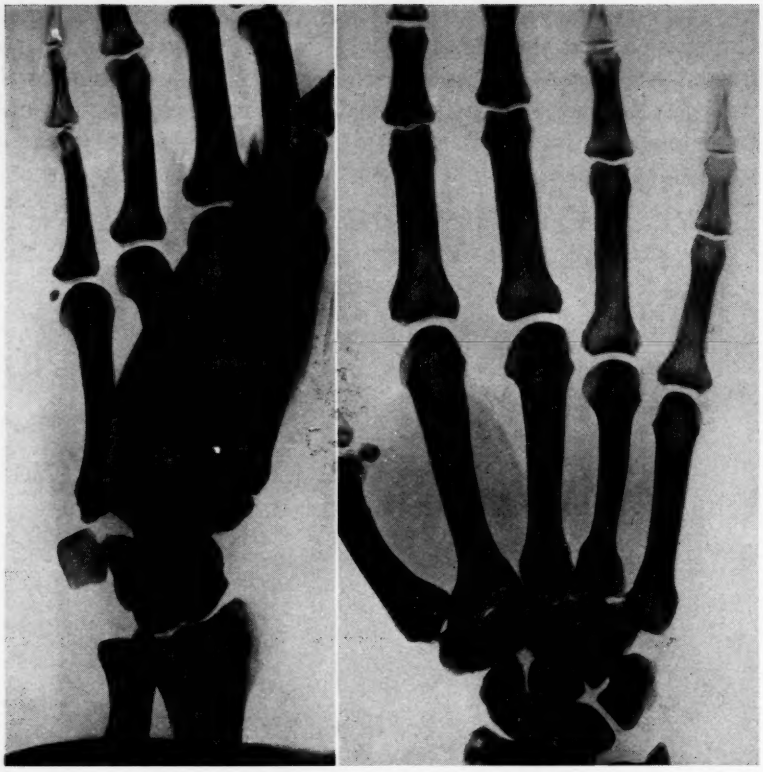


FIG. 387.—*Case* 3. Dislocation of fifth metacarpal bone: similar displacement to *Cases* 1 and 2.

FIG. 388.—*Case* 4. Proximal end of fifth metacarpal dislocated forward into palm of hand.

two weeks before he was admitted to hospital. Radiographs showed a marked forward and outward displacement of the base of the fifth metacarpal. This case is evidently an exact parallel to the fourth case in the present series. Attempts at manipulation failed entirely to alter the position of the displaced metacarpal and consequently open operation was undertaken. An extensive dissection of the palm was necessary to free the bone, but it was successfully replaced and remained stably reduced. Good functional recovery occurred in a few weeks.

It seems probable that in cases of this type with extreme displacement of the base of the metacarpal across the palm a closed reduction would be difficult or impossible. Open operation may therefore be resorted to, although it appears to be entirely unnecessary in the other cases in which the displacement is to the inner side and is less extreme.

SUMMARY

1. Four cases of isolated dislocations of the base of the fifth metacarpal are described. One other case has been previously recorded.

2. Two types of displacement are exemplified by the present series; one an extreme degree of outward displacement and the other a less extreme inward and forward displacement.

3. Reduction of the latter group should be easy, but a long period of continuous traction is necessary to maintain the corrected position.

4. Open operation may be necessary for those cases in which the metacarpal is displaced outwards across the palm.

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RUPTURE OF THE LONG HEAD OF THE BICEPS BRACHIALIS WITH NOTES ON FOUR CASES

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THE four cases of rupture of the long head of the biceps which form the subject of this paper were all seen between June, 1934, and February, 1935. Nevertheless it is an uncommon injury. Thus Harry Platt,[1] writing in 1931 on ruptures of tendons generally, said that he had never seen a case. In 1925 Gilcreest,[2] in an exhaustive paper, pointed out that little had been written on the subject in English but that there were several lengthy papers in the French and German literature. Loos[3] in 1900 had collected 62 published cases, to which he added 11 more. By 1927 McKenna[4] found 81 cases in the literature, including those of Loos, and he himself added 1 more. He pointed out that of the 81 cases, only 15 had come to operation. Rankin[5] in 1933 described 4 further cases. It would appear, therefore, that fewer than 100 cases have so far been published and that hitherto no writer in this country has recorded his experience of the condition.

Etiology.—In the writer's four cases the patients were all men, their ages varying between 51 and 92, and in each case the right arm was involved. The patients were all lean, wiry subjects with arteriosclerotic changes in the peripheral blood-vessels. In one the Kahn reaction was strongly positive, and in this case the rupture occurred from direct injury to the biceps, although the violence was not applied at the point of rupture; in another case the injury occurred in a motor accident and the mechanism of its production could not be ascertained. In the two remaining cases indirect violence was the cause. It is probable that there is always an osteo-arthritis of the shoulder and that this is a predisposing cause of the condition.

Diagnosis.—Rupture of the long head of the biceps produces a characteristic syndrome, and the diagnosis should never be difficult. The patient complains of a sudden acute pain in the upper limb, usually in the region of the shoulder-joint, and he notices thereafter that the arm is weak. The pain is aggravated on voluntary flexion of the elbow, and supination of the forearm is also painful. On telling the patient to flex the elbow, one finds that the lateral half of the belly of the biceps contracts into a firm circumscribed mass, about the size of a tangerine orange. This mass is drawn down towards the elbow-joint and one feels an 'emptiness' above it. In the two cases seen within a few days of the accident it was possible to palpate the tendon of the long head lying coiled up in this space and it felt like a worm lying just beneath the skin. If the case is not seen until some months after the injury, this 'coiled worm' sensation is not noted, for the effusion organizes into fibrous tissue which binds the coils of the tendon together and they thus lose their identity. In some cases the coiled tendon is not felt for the reason that the ruptured portion remains in the bicipital groove.

Anatomical Considerations.—In some cases the tendon gives way at its origin from the rim of the glenoid cavity—that is, the injury is a true avulsion. In other cases the rupture occurs at the upper end of the bicipital groove, and here it would appear that a ridge of osteophytes running between the tuberosities of the head of the humerus causes friction which leads to flattening and 'fraying' of the tendon—that is, the injury is a rupture of the tendon in its continuity. When the rupture takes place at the glenoid rim it sometimes happens that the tendon is not drawn out of the bicipital groove into the arm. If such a case is untreated at the time, the tendon will then take up a new, but unstable, attachment in the groove. This is a most important practical point, for when one comes to explore the biceps at operation the muscle appears to be intact and it may seem, at first sight, and unless one is aware of this contingency, that the diagnosis is in error. In these cases the tendon must be dragged away from its new attachment in order to deliver it into the wound.

Treatment.—To restore full strength and function to the limb it is manifest that the treatment must be operative. To leave so large a portion of the biceps with an absent or unstable attachment must inevitably weaken the arm to an extent that will almost certainly prevent a manual worker from carrying out his former employment. In old people, and those whose occupation is sedentary, operation need not be insisted upon, and this applies particularly to those cases in which a new attachment has been acquired in the bicipital groove.

A restoration of the normal anatomy of the biceps is clearly unattainable when the tendon has been avulsed from its origin. When the rupture has taken place in the bicipital groove, direct suture is equally impracticable, for in these cases the ends of the tendon are frayed and thinned.

The long head of the biceps is, to a certain extent, an abductor of the arm. This, however, is a subsidiary part of its function and may be ignored. The important consideration is to restore its power of flexing the elbow and supinating the forearm. This may be attained by suturing the tendon in such manner that it will reinforce the action of the short head of the muscle.

The writer's friend and teacher, the late W. H. Trethowan, was probably the first to solve the problem. In 1921 he operated upon a case in which the diagnosis of avulsion of the long head of the biceps had not been established beforehand. The injury was of some months' duration and the clinical evidence suggested that the rupture had taken place either in the belly of the muscle or at the junction of the belly with the tendon. Trethowan solved the problem which confronted him in his customary inspired fashion by attaching the ruptured tendon to the coracoid process of the scapula, having first passed it through the origin of the short head. The steps of this operation are as follows:—

A long incision is made, beginning at a point medial to the coracoid process, just below the clavicle. The incision passes laterally for about 3 in., whence it curves downwards, in the long axis of the humerus, to a point 2 or 3 in. above the elbow-joint. The triangular flap of skin thus mapped out is reflected medially and the biceps is now exposed and its condition investigated. If the tendon is not found lying coiled up in the arm, the space between the deltoid and the insertion of the pectoralis major is dissected free and the deltoid is retracted laterally. The tendon of the long head is now exposed, and with a sharp jerk it may be pulled free from the new attachment which it may have acquired in the bicipital

groove. The coracoid process is next identified lying deeply between the deltoid and the clavicular head of the pectoralis major. The periosteum over the coracoid is split vertically and elevated, and a small incision is made through the origin of the short head of the biceps. A long Mayo-Oschner forceps is now thrust from before backwards through this incision, and it is made to pass beneath the insertion of the pectoralis major, and anterior to the teres muscles, so that it emerges in the middle of the arm to the lateral side of the belly of the short head. The end of the ruptured tendon is seized in the forceps and pulled upwards through the origin of the short head, to the belly of which it will now come to lie parallel and slightly posterior. The tendon of the long head is sutured to the periosteum of the coracoid process, two or three stitches also being placed to attach it to the origin of the short head, through which it is passing. Unabsorbable material should be used for the sutures, the writer having a personal preference for silkworm-gut. Before suturing the tendon in its new position it is essential that it should be pulled as taut as possible, with the elbow flexed and the forearm supinated and lying well in front of the chest. In this position the new origin and the insertion of the long head are approximated towards one another, and it is attained by fixing the hand of the injured side to the opposite shoulder. This position is maintained until the removal of the stitches at the end of twelve days. For the next fortnight a 'collar-and-cuff' bandage is worn, and during this time the patient is encouraged to perform active contractions of the biceps. After this a sling, with the elbow at a right angle, is worn for a few days, and then full extension of the elbow, in which gravity assists, is gradually allowed and active movements of the shoulder-joint in all directions are encouraged.

Ludington[6] has pointed out that the following alternative methods have been suggested: (1) Suture of the ruptured tendon to the pectoralis major (Hoffmann); (2) Suture in the sulcus intertubercularis (Roloff); (3) Suture to the short head of the biceps (Bazy); (4) Suture to the coracoid process of the scapula (Perthes). Colton and Morrison[7] have recently advocated (5) Looping the torn tendon round the insertion of the deltoid. The first and fifth methods would clearly yield poor anatomical and functional results, while the second method introduces considerable technical difficulties. It will be seen that the method the writer has described, which is to be attributed to Trethowan, is a combination of the third and fourth methods. There is no doubt that it yields excellent results, it is not difficult to perform, and, as one patient has expressed it, his "arm feels quite as strong as it did before the accident"—this statement being borne out by the fact that he is able to crank the engine of a heavy motor lorry which has become stiff through standing all night in cold weather.

CASE REPORTS

Case 1.—A. C., male, aged 51, omnibus overseer. Referred to the writer on June 21, 1934, by Dr. S. Allman Hall, of Dunmow. This patient had been cranking a fire-engine a few days before, when the engine back-fired and he experienced sudden sharp pain in the right upper arm and shoulder region. ("The engine was very tight, and as I came to the top of the swing it back-fired and forced my arm down straight."). The arm felt very weak and he was unable to use it for anything demanding even moderate strength. On examination the diagnosis of rupture of the long head of the biceps was quite obvious. On voluntary flexion of the elbow the lateral part of the belly of the biceps contracted into a firm circumscribed mass, and just above this the tendon could be felt lying coiled up in the characteristic

manner described above. There was little or no discoloration or swelling from effused blood. At operation on June 28 it was evident that the tendon had torn out from its origin at the glenoid rim. The operation described above was carried out and progress was uneventful. Three months later voluntary power in the biceps was normal and the movements of the shoulder- and elbow-joints were complete. The only abnormality to be detected was that on contraction the whole of the belly of the biceps appeared to have been displaced slightly inwards, this appearance being accounted for by the new direction of the muscle fibres.

Case 2.—W. W., male, aged 65, labourer. Referred to the writer by Dr. J. H. Hall, of Maldon. Six weeks previously the patient had been involved in a motor collision. On examination the characteristic signs of rupture of the long head of the biceps were evident. It was not possible to elucidate the precise mechanism of the rupture. This man's general health was not satisfactory—he had extremely tortuous, hard arteries, with a blood-pressure of 246/150, and chronic emphysema. In view of this, and since his work was not heavy and he expressed himself himself as able to do it, operation was considered inadvisable.

Case 3.—E. B., male, aged 60, farm worker. Referred to the writer by Dr. R. C. Little, of Witham. Eight months previously the patient had been lifting a full bucket of milk from beneath a cow with his elbow flexed (i.e., the biceps strongly contracted), when the cow kicked forwards with its hind leg and its hoof struck the middle of the biceps. There was a good deal of discoloration of the skin from effused blood, but the precise nature of the injury was evidently not diagnosed at the time. The arm was rested for four weeks and the man then resumed his occupation. He remained at work for six months, when pain and weakness in the arm compelled him to give up. On clinical examination the lateral half of the belly of the biceps contracted into a firm rounded mass above which the characteristic 'emptiness' was felt, but the tendon was not palpable. At operation the tendon of the long head was seen in its normal position, and it was clear that the ruptured tendon had taken up a new attachment in the bicipital groove. The tendon was jerked away from this adventitious attachment and sutured to the coracoid process in the manner described above.

Case 4.—A. M., male, aged 92, retired Army coach. This patient, an extremely hale and hearty old gentleman, had been a well-known athlete in his younger days, and even at his present great age it is his invariable custom to walk four miles every day in all weathers. The rupture of his biceps was caused by reaching suddenly for something above his head. When seen a few hours later, all the characteristic signs were present, including the 'coiled worm' sensation in the gap above the retracted belly of the long head. In view of his age, operation was not advised.

SUMMARY

1. The literature of rupture of the long head of the biceps is briefly reviewed.
2. The clinical features and etiology of the condition are considered.
3. An operation for the repair of the ruptured tendon, together with its appropriate after-treatment, is described in detail.
4. Notes of four cases of the injury are appended.

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PROGRESSIVE POST-OPERATIVE CUTANEOUS GANGRENE

By H. T. COX

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THE facts herewith recorded relate to a case of progressive post-operative cutaneous gangrene developing in a patient operated on for a perforated duodenal ulcer. This case is presumably the fourth of its kind to be reported in Great Britain.[1]

The patient, a man of 44 years, was admitted to Withington Municipal Hospital on Jan. 8, 1935, with an acute abdominal crisis of nine hours' duration. On examination the symptoms and signs presented were those of an acute perforated peptic ulcer. Immediate laparotomy was performed and the diagnosis confirmed, the ulcer being present on the anterior wall of the first part of the duodenum. This was sutured in the usual way and the fluid in the peritoneal cavity partially evacuated. Closure of the abdominal wall was carried out in layers with the addition of silkworm-gut tension sutures and a small rubber drain in the lower end.

The immediate post-operative period was uneventful. Sepsis, however, became evident on the seventh day as shown by a profuse discharge of pus from the lower end of the wound. The tension sutures were removed on the twelfth day. The localized discharge persisted until the fourteenth day, when wound infection became general. There was pain, tenderness, redness, and induration around the whole extent of the abdominal incision. This generalized inflammation then subsided, leaving a small persistent area at the site of the skin puncture which marked the point of emergence of the upper tension suture, i.e., on the left side of the incision. Inflammation at this point slowly but steadily progressed. A purulent ulcer developed with a narrow black band of gangrene at its margin. On the outer side of this band lay a zone of tender induration with an ill-defined edge; on the inner side there appeared a white slough rapidly assuming a dirty greenish colour.

As the lesion increased in size the central slough became detached in portions, revealing healthy, red, granulating tissue. This unusual lesion therefore presented very definite characteristics. Four zones were readily recognizable: an outer zone of tender induration, bluish in colour, with an ill-defined edge and about 1 in. in width; a second zone of black gangrene, about $\frac{1}{2}$ cm. in width, with sharply demarcated edges, situated at the spreading margin of the ulcer; on the base of the ulcer two more areas, the slough originally white but now of a dirty green colour, and, where this had separated, healthy, red, granulating tissue.

The temperature ran an even course with an occasional short bout of pyrexia up to 100° F. No enlargement of the axillary or inguinal glands was detected. All local therapeutics failed to arrest the spread of infection. The lesion continued

to progress with the maximum area of spread on the left lateral side; its right lateral margin never transgressed the scar of the original incision.

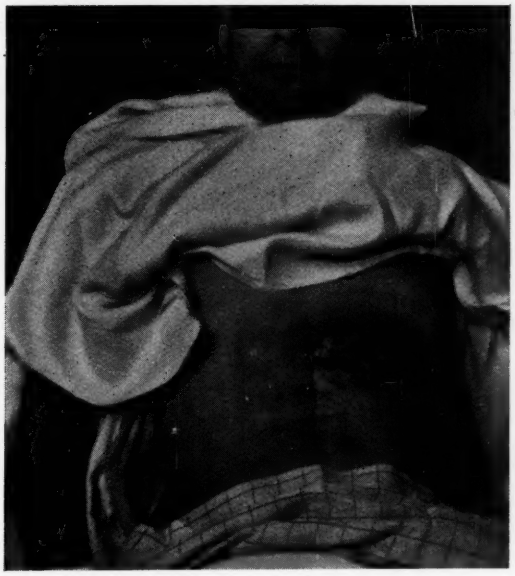

FIG. 389.—Photograph taken three days prior to excision.

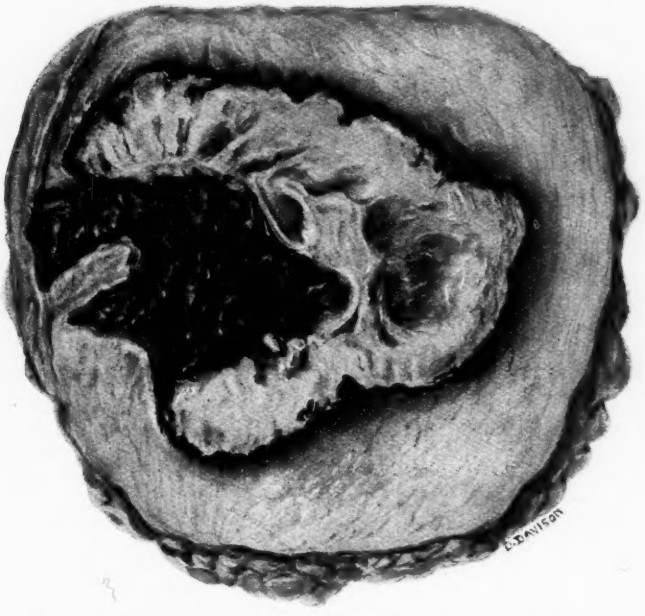

FIG. 390.—Drawing of specimen of progressive post-operative cutaneous gangrene after excision.

On Feb. 3, twenty-six days after operation, when the ulcer measured $3\frac{1}{2}$ by $2\frac{1}{2}$ in., a diagnosis of progressive post-operative cutaneous gangrene was made. The ulcer was covered with gauze, sealed with collodion, and widely excised with

a scalpel under intravenous evipan anæsthesia. The tissues removed included the ulcer with 1 in. of surrounding skin on the left lateral side, 1 cm. on the right lateral side, and all subcutaneous tissues down to the aponeurosis. *Fig.* 389 is a photograph of the condition taken three days before excision, and *Fig.* 390 is a drawing of the specimen after excision.

The patient refused a skin-graft, but the wound had completely epithelialized three months after excision.

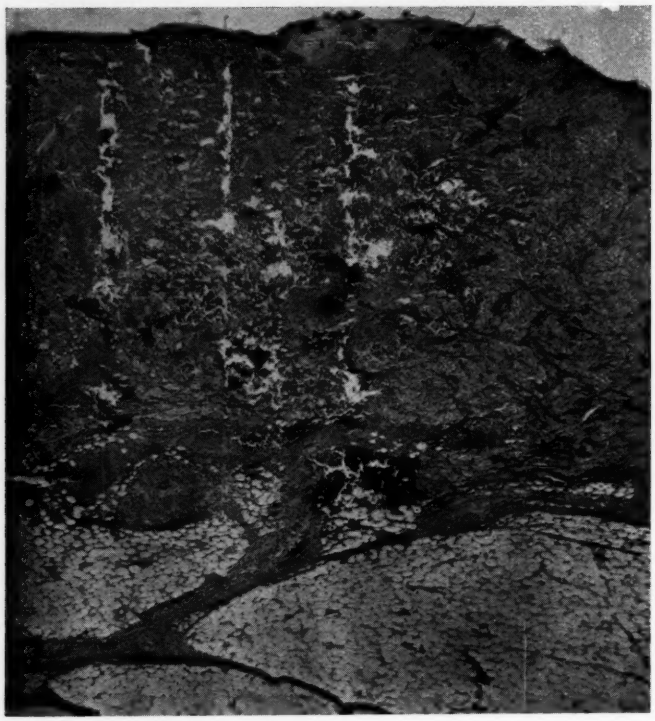

FIG. 391.—Section through spreading edge showing limitation of the infection to the epidermis and true skin. (× 15.)

PATHOLOGICAL REPORT.—I am indebted to Dr. Dawson for the following pathological reports :—

Pus from Ulcer.—Microscopically and on cultivation. *Streptococcus pyogenes* was present in large numbers.

Blood-count.—

White blood-cells	16,100	
Polymorphs	79	per cent
Lymphocytes	18	,,
Monocytes	3	,,
Eosinophils	–	
Basophils	–	

Wassermann Reaction.—Negative.

Pus from Ulcer after Excision.—A non-hæmolytic streptococcus was isolated which grew indifferently under aerobic and strict anaerobic conditions. A heavy growth in peptone broth was inoculated subcutaneously into a guinea-pig and did not cause a general infection or local inflammation.

Section of Ulcer through the Spreading Edge (*Fig.* 391).—Histological examination showed necrosis of epidermis and dense leucocytic infiltration of the corium with early slough formation. The underlying subcutaneous tissues are relatively uninvolved.

The reasons for presenting this case as an example of progressive post-operative cutaneous gangrene are: (1) The presence of a definite zone of black gangrene; (2) Microscopic evidence that the spreading margin is limited to the true skin; (3) The failure of local therapeutics; and (4) That it occurred after operation.

I wish to express my thanks to Dr. Gamble, Medical Superintendent, and Mr. Holt, Consulting Surgeon, for permission to report this case.

REFERENCES

[1] NIGHTINGALE and BOWDEN, *Brit. Jour. Surg.*, 1934, xxii, 392.
[2] STEWART and WALLACE, *Ibid.*, 1935, xxii, 642.

INTRAVENOUS PYELOGRAPHY IN A SERIES OF CASES AFTER TRANSPLANTATION OF THE URETERS

By G. GREY TURNER, London
and JAMES H. SAINT, Newcastle-upon-Tyne

The condition of patients in whom the ureters have been transplanted into the sigmoid colon presents two aspects of considerable interest and importance. The first concerns the pathological changes which may take place in the upper urinary tract and their effect upon renal function, and the second, the rôle played by the large bowel in the storage of urine.

Certain clinical observations, pertinent to the first aspect, were made by the senior author in his study of the after-history of a series of 17 personal cases published in this Journal in 1929.[1] They were as follows: "Judged by the standard of the general health, it may be said that none of the patients in this series shows definite evidence of gross renal insufficiency. At the outset I must say I feel convinced that most of these patients develop some degree of ascending renal infection. This is borne out by the fact that in the two cases in which it has been possible to make post-mortem investigations there has been definite and gross evidence of its existence, in spite of the fact that during life neither of these patients suffered from symptoms indicating its presence."

Because several patients of the series were in good health at the time of publication of the paper though they had previously suffered on various occasions from symptoms indicating renal infection, it was further stated that: "From these clinical observations it would appear that a moderate degree of renal infection is not inconsistent with average good health and well-being."

For more exact information regarding pathological changes which may have taken place in the upper urinary tract, we have depended until recent years upon ascending pyelograms, but it would be difficult, as well as highly undesirable, to ascertain in this way information regarding kidneys the ureters of which have been transplanted into the colon. It is natural, therefore, that for use in such cases we should acclaim and hasten to take advantage of the advent of *uroselectan*, the iodine-compound dye which is opaque to X rays and which, after injection into the circulation, is excreted by the kidneys and thus not only serves the same purpose as ascending pyelography but also, by the degree of the density of its shadow, gives some indication of the functional activities of those organs.

The fact that not only the rectum but the entire colon might act as a reservoir for the urine was mentioned in the publication referred to above, when it was stated: "The problem of where the urine is actually stored in the bowel is of great interest. It cannot be in the rectum, for this part of the bowel is quite unable anatomically to contain the quantity of fluid which sometimes collects. . . . Post-mortem specimens showing enlargement and distension of the colon suggest that the whole of the large bowel may act as the reservoir."

We thought that uroselectan might also be able to contribute to the solution of this problem, for it was felt that the shadow shown by the dye would demonstrate the level to which the urine had risen in the rectum or colon. It was obviously necessary for our purpose, however, that the quantity of urine in the bowel, when taking the X-ray, should be as great in amount as possible, and we therefore decided to allow six hours to elapse after the injection of the dye, no evacuation of the bowels taking place in the meantime.

The investigation was undertaken in six cases, in all of which the operations had been performed by the senior author at the Royal Victoria Infirmary, Newcastle-upon-Tyne, while he was a member of the honorary staff of that institution. Four of them, all subjects of congenital defects of the bladder and urethra, were reported in full by him in the publication to which reference has already been made. They are the first four the results of whose investigations are described below, and the numbers in parentheses are those which they bore when reported on that occasion. The remaining two cases were operated upon at a later date; in one the condition present was ectopia vesicæ, and in the other vesico-vaginal fistula following injuries during parturition.

All the cases were admitted to hospital for this investigation. The patient was given an ounce of castor oil in the afternoon of the day previous to the investigation and allowed only a light supper. No further food was partaken of until after the completion of the investigation. The bowels were evacuated shortly before 9 a.m., when the dye was injected into the median basilic vein with the usual precautions and technique for such a procedure. Uroselectan B was used, it being the outcome of further research by von Lichtenberg and Binz, the discoverers of uroselectan itself, and possessing three distinct advantages over the original compound in that (1) it is made up in ampoules ready for use, (2) only 20 c.c. need be injected in contrast to the 100 c.c. of uroselectan, and (3) it is much more certain in its results. Once the dye was injected, the taking of fluids was discouraged, for it was felt that they might augment the amount of urine to such an extent as to render necessary evacuation of the bowels before six hours had elapsed and so defeat the object of the second part of the investigation. X-ray films of the abdomen were taken by a portable apparatus at the following intervals after injection: (1) 5 minutes, (2) ½ hour, (3) 1½ hours, and (4) 6 hours.

A brief history of each case is given in the following pages, together with a description of the uroselectan films and the latest available report on the condition of the patient.

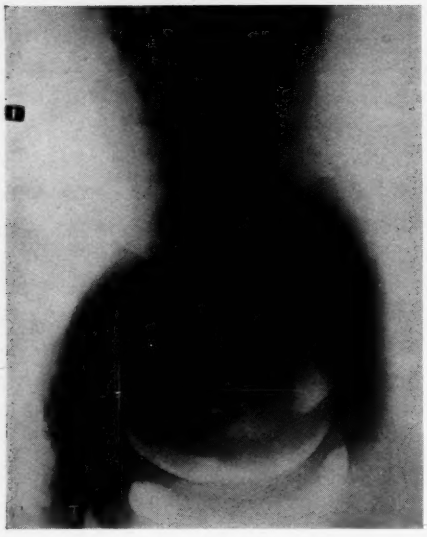

FIG. 392.

FIG. 393.

FIG. 394.

FIG. 395.

FIGS. 392-395.—*Case* 1. Uroselectan films taken 5 minutes, ½ hour, 1½ hours, and 6 hours respectively after injection of the dye.

CASE REPORTS

Case 1 (No. 2).—Mrs. E. S. (*Fig.* 416), aged 41 at time of uroselectan investigation, April 21, 1933.

HISTORY.—Ureters transplanted in November and December, 1914, on account of epispadias with incontinence, the patient then being 22 years of age. Uninterrupted recovery from both operations and has enjoyed very good health since. Married in November, 1916. First child born in 1920 and a second in 1922. Patient about seven months pregnant when admitted for investigation.

UROSELECTAN FILMS (*Figs.* 392–395).—

Fig. 392: Right—only one (dilated) calix seen.
Left—normal pelvis and lower calices of kidney are seen. The upper 3 in. of the ureter is visible and shows slight dilatation.
This film was taken at such a level as to cut off the upper poles of the kidneys.

Fig. 393: Right—shadow of kidney plainly visible; all calices show dilatation, this being especially well-marked in those of the upper pole. No ureter seen.
Left—kidney shadow has disappeared. About another inch of ureter is seen just below that portion on *Fig.* 392 and shows slight dilatation.
There is a faint shadow in front of the sacrum.

Fig. 394: Right—the calices are shown much as in *Fig.* 393, but the shadow is of slightly greater density. The pelvis of the kidney and the whole of the ureter above the brim of the pelvis are visible and are much dilated.
Left—no kidney shadow seen. The ureter is shown much the same as in *Fig.* 393.
The density of the shadow in the pelvis has increased to such an extent as practically to obliterate the outline of the fœtal head.

Fig. 395: Right—faint shadow of upper calices and ureter still seen.
Left—no kidney or ureteric shadow present.
The shadow in the pelvis is very dense and has extended so as nearly to fill it. The iliac, descending, and transverse portions of the colon are not outlined, but the dye is present in good concentration in the cæcum and ascending colon as far as the hepatic flexure.

The blood-urea was 35 mgrm. per 100 c.c.

SUMMARY OF CONDITION OF KIDNEYS (19½ years after transplantation of both ureters).—On the right side the kidney is the seat of a well-marked hydronephrosis and the ureter is also much dilated. The failure of the dye to give a good shadow in the first film indicates some impairment of the renal excretory function. The obstruction present is of sufficient degree to cause retention of the dye in the kidney and ureter for 6 hours. The left kidney appears normal both as regards structure and function, but the ureter is slightly dilated, indicating that a mild degree of obstruction is present.

LATEST REPORT (April 10, 1935).—The patient states that her general health is good. While the length of time between evacuations of the bowels varies somewhat, the average is about six hours. The longest interval was from 5.30 p.m. to 9.0 a.m. the following morning. Her third child was born on June 24, 1933, about two months after her investigation. The confinement was normal, although she thinks the pain was greater than on the last occasion eleven years previously. The baby was normal and has progressed satisfactorily and the mother made a splendid recovery from the confinement.

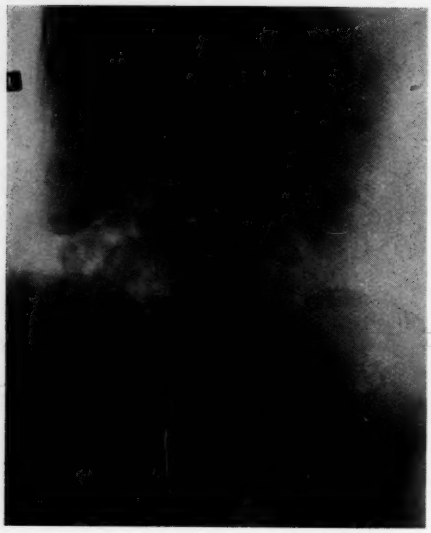

FIG. 396.

FIG. 397.

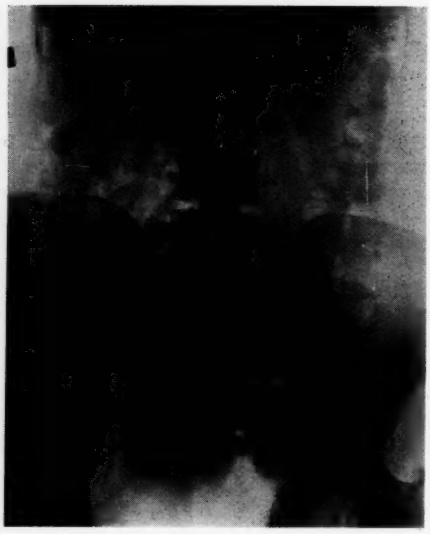

FIG. 398.

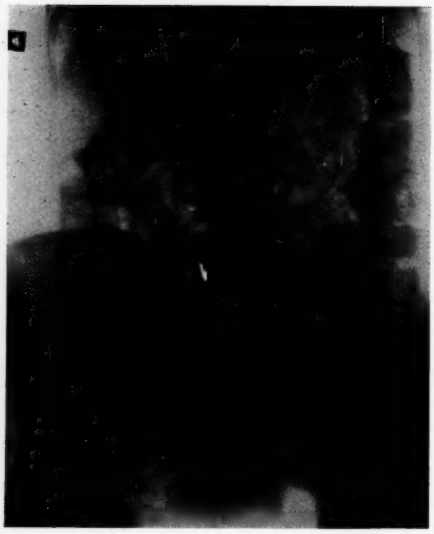

FIG. 399.

FIGS. 396–399.—*Case* 2. Uroselectan films taken 5 minutes, ½ hour, 1½ hours, and 6 hours respectively after injection of the dye.

Case 2 (No. 5).—V. K. (*Fig.* 417), aged 20 at time of uroselectan investigation, Nov. 30, 1931.

HISTORY.—The ureters were transplanted in August and September, 1917, because of ectopia vesicæ, the patient being then 6 years old. Following the transplantation of the second (right) ureter, her condition began to give rise to anxiety, vomiting, abdominal distension and loss of weight occurring together with a slight evening rise of temperature. The abdominal wound was opened with sinus forceps and about 10 oz. of pus evacuated; two weeks later a further abscess was found in the abdominal incision. Under the anæsthetic a mass was discovered in the right loin; another incision was made close to the iliac crest and a retroperitoneal abscess evacuated. After a short period during which the pulse and temperature were elevated, the patient gradually improved and was soon able to leave hospital. The mucous membrane of the bladder was removed in April, 1921. She has enjoyed excellent health and has developed into a fine, strong, healthy, young woman. An X-ray of the urinary tract taken before the injection of uroselectan showed two small shadows in the right renal area suggestive of calculi.

UROSELECTAN FILMS (*Figs.* 396–399).—

Fig. 396: Right—a few faint shadows, evidently those of the upper calices, are seen above those of the calculi.
Left—shadow of normal kidney pelvis and calices. The ureter is shown in most of its course and is slightly dilated in its lower third.

Fig. 397: Right—shadows increased in size and density, showing somewhat dilated calices.
Left—kidney pelvis and calices represented by a fainter shadow than in *Fig.* 396. Ureteric shadow has almost disappeared.
A small, fairly dense shadow is seen in front of the lower part of the sacrum.

Fig. 398: Right—further increase in density, but not in size, of shadows in renal area.
Left—very faint shadow of kidney pelvis and calices.
The shadow in front of the sacrum has increased in size and density and has spread to the left, evidently due to the dye in the lower part of the pelvic colon.

Fig. 399: Both renal and ureteric shadows have disappeared.
The large bowel is outlined by a shadow of good density from the rectum to the cæcum, similar in appearance to a barium enema, except for the greater density of the latter.

The blood-urea was 46 mgrm. per 100 c.c.

SUMMARY OF CONDITION OF KIDNEYS (14 years and 2 months after transplantation of both ureters). The right kidney contains two or three calculi, and a mild degree of hydronephrosis is present. The shadows shown by the dye indicate that the function of this kidney is somewhat impaired. On the left side, although there is a slight dilatation of the ureter indicating a mild degree of obstruction, the kidney possesses normal structure and function.

FURTHER HISTORY.—Three small calculi were removed from the right kidney in December, 1932. The operation was followed by the development of a persistent sinus and the patient was re-admitted for operation in November, 1933. On this occasion another small stone was removed and after three weeks the wound healed, in which condition it has remained.

LATEST REPORT (April 12, 1935).—The patient writes to say that she is perfectly well. Evacuation of the bowels takes place, on the average, six times during the twenty-four hours, but on one occasion eleven hours elapsed between evacuations.

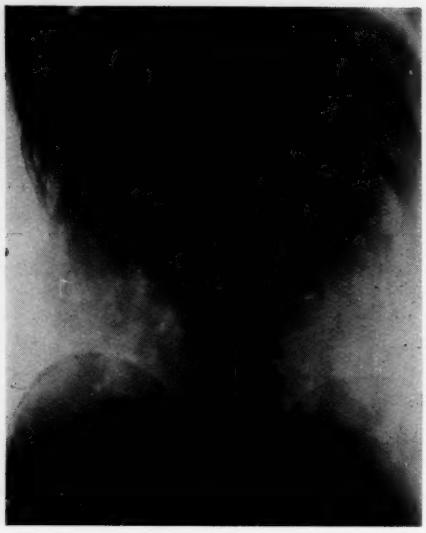

FIG. 400.

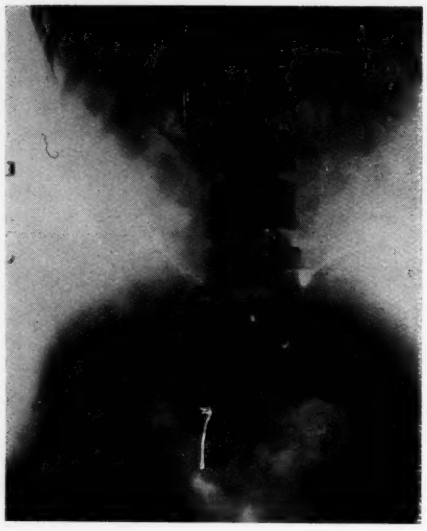

FIG. 401.

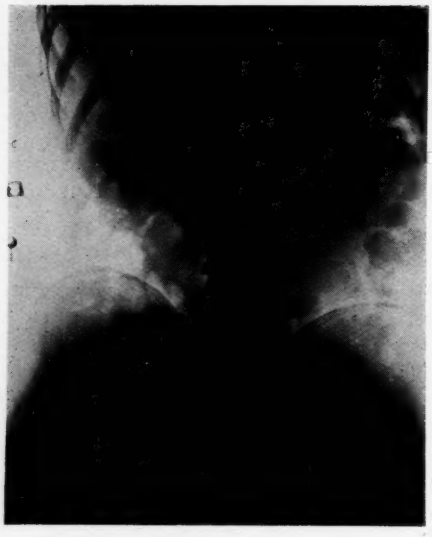

FIG. 402.

FIG. 403.

FIGS. 400–403.—*Case* 3. Uroselectan films taken 5 minutes, ½ hour, 1½ hours, and 6 hours respectively after injection of the dye.

Case 3 (No. 7).—W. P., aged 18 at the time of uroselectan investigation, Nov. 28, 1932.

HISTORY.—The patient suffered from complete epispadias with incontinence. The right ureter was transplanted in August, 1918, the patient then being 5 years of age. After the operation there developed what appeared to be a pelvic peritonitis. Under anæsthesia the lower sutures of the abdominal wound were removed, and as soon as the peritoneal cavity was opened, there was a rush of offensive urine followed by pus. Drainage was provided and the patient steadily improved, being soon well enough to return home. The sinus closed within a few days of his leaving hospital. The left ureter was transplanted in May, 1920. On the tenth day following this operation there was a slight urinary leakage from the site of the abdominal drainage tube; it continued for a week, when it ceased spontaneously, the remainder of his convalescence being uneventful. The intervening health has been very good.

UROSELECTAN FILMS (*Figs.* 400–403).—

Fig. 400: Right—kidney shadow large but faint and blurred, indicating an apparent hydronephrosis.
Left—kidney shadow more dense and distinct than on right side and not showing any dilatation.

Fig. 401: Both kidney shadows show increased density.
Right—the dilated calices are now well defined, showing definite hydronephrosis.
Left—the calices are shown better than on the previous film; clubbing is seen but there is no dilatation.
There is a faint shadow at the level of the coccyx.

Fig. 402: Right—the kidney shadow shows increased density; the ureter is outlined clearly down to the superior border of the sacro-iliac joint and is uniformly and widely dilated.
Left—the density of the kidney shadow is much the same as in the previous film but is somewhat obscured by gas shadows.
The size of the shadow in front of the sacrum is increased and dye is present in parts of the iliac and descending colon.

Fig. 403: Both kidney shadows have disappeared.
Judged by the density of the shadow, the dye is present in good concentration in the cæcum, ascending colon, right third of transverse colon, and the upper portion of the descending colon.

The blood-urea was 48 mgrm. per 100 c.c.

SUMMARY OF CONDITION OF KIDNEYS (14 years and 3 months after transplantation of both ureters).—On the right side there is a well-marked hydronephrosis and hydro-ureter with some impairment of kidney function. On the left side the clubbing of the calices indicates an incipient stage of hydronephrosis. The function of the kidney is good although the degree of obstruction present is sufficient to cause enough dye retention to result in an increased density of shadow up to one and a half hours.

LATEST REPORT (April 10, 1935).—The patient states that he is enjoying very good health. He finds that a bowel movement is generally necessary four times during the day and twice at night, but he has known as long as eight hours elapse between evacuations.

FIG. 404.

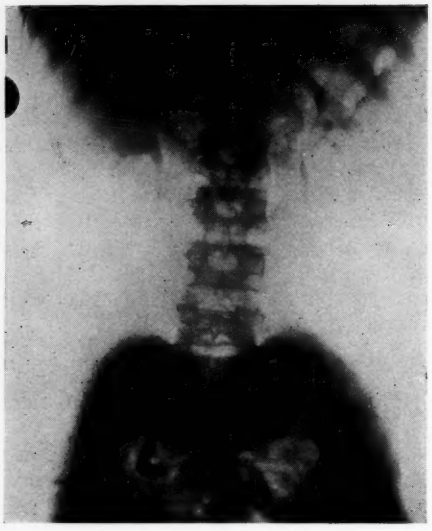

FIG. 405.

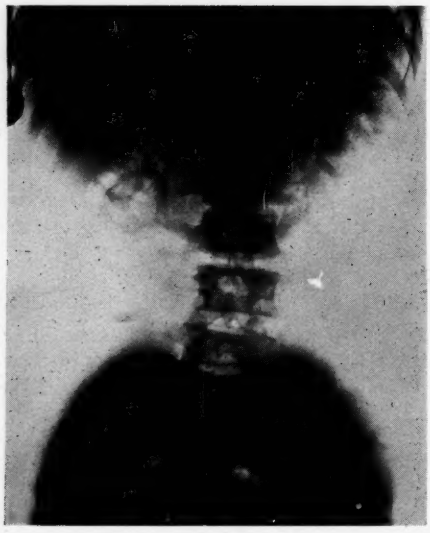

FIG. 406.

FIG. 407.

FIGS. 404–407.—*Case* 4. Uroselectan films taken 5 minutes, ½ hour, 1½ hours, and 6 hours respectively after injection of the dye.

Case 4 (No. 12).—J. P., aged 19 at time of uroselectan investigation, April 10, 1933.

HISTORY.—Both ureters transplanted in October, 1924, for ectopia vesicæ, the patient being 10 years old. The left ureter was transplanted first and an interval of three weeks allowed to elapse before the right ureter was dealt with in a similar manner. Except for a slight setback soon after the first operation, apparently due to an attack of renal infection which quickly passed off, the patient recovered well from both operations and has enjoyed good health ever since.

UROSELECTAN FILMS (*Figs.* 404–407).—

Fig. 404: The pelves and calices of both kidneys are clearly shown by shadows of good density; they are normal, showing neither clubbing nor dilatation.
Right—the upper portion of the ureter is shown and is not dilated; in the pelvis, just below the sacro-iliac joint a small part of the ureter is seen and is dilated.
Left—that portion of the ureter above the sacro-iliac joint is very faintly outlined and shows slight dilatation; just below this joint is outlined a small, dilated portion of the ureter turning inwards above the ischial spine.
Low in the pelvis, between the separated pubic bones, a shadow has already made its appearance.

Fig. 405: Both kidney shadows are less dense.
Right—the outline of the ureter is seen both above and below the sacro-iliac joint; above, it is slightly dilated, while below, the dilatation is more marked and the ureter is seen to bend inwards.
Left—the ureter is seen only above the crest of the ilium, and, in the middle of this part of its course, shows a slight dilatation.
The shadow in the pelvis has spread upwards and has now reached the level of the lower part of the sacrum.

Fig. 406: Both kidney shadows are of diminished density and the calices are not so well outlined. Only the upper portions of the ureters may be discerned and with difficulty. The shadow in the pelvis has increased in both size and density.

Fig. 407: Both kidney and ureteric shadows have disappeared. The shadow in the pelvis is not so large or dense as in the last film and no dye is present in the colon.

NOTE.—This boy stated emphatically that he could not allow more than four hours to elapse between evacuations. Because of this, he was instructed to empty his rectum immediately after the third film so as to allow about four hours for further accumulation before the taking of the last film. However, the poor result of the last film shows clearly that almost all the dye had been excreted within one and a half hours of its injection. The details of the ureteric shadows described above, while seen in the original films, are not shown in the photographs.

The blood-urea was 46 mgrm. per 100 c.c.

SUMMARY OF CONDITION OF KIDNEYS (8½ years after transplantation of both ureters).—Both kidneys show a normal structure. While both ureters show some degree of dilatation, the obstruction causing this has not been sufficient to affect the kidneys up to the time of investigation. The manner in which the dye was excreted by the kidneys indicates that each possesses an unimpaired function. It is probable that this case represents as perfect a result as it is possible to obtain.

LATEST REPORT (April 13, 1935).—The patient writes to say his general health is good. He has to empty his rectum about every two hours during the day and generally twice during the night. There is occasional nocturnal incontinence.

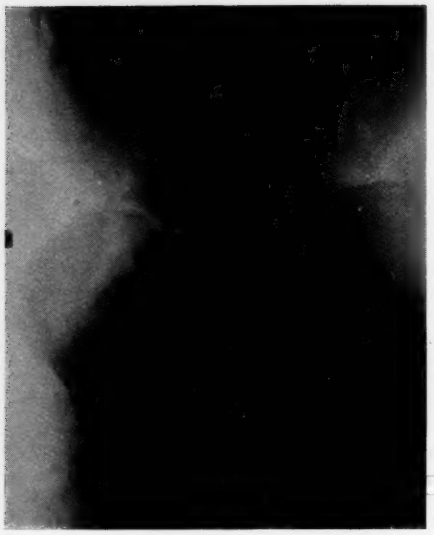

FIG. 408.

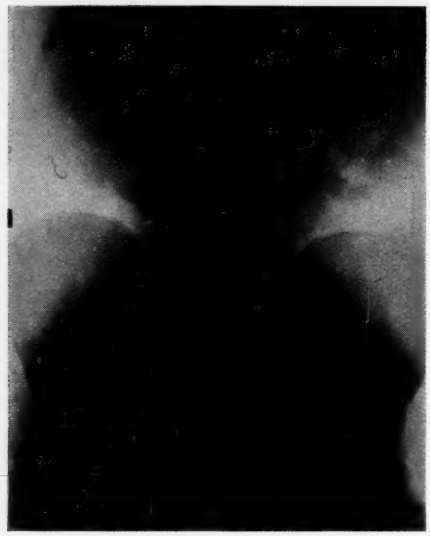

FIG. 409.

FIG. 410.

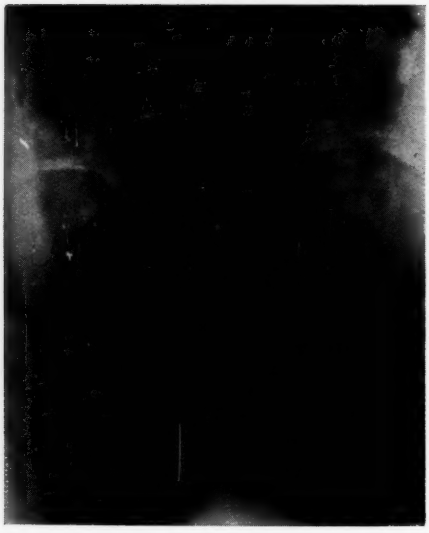

FIG. 411.

FIGS. 408–411.—*Case* 5. Uroselectan films taken 5 minutes, ½ hour, 1½ hours, and 6 hours respectively after injection of the dye.

Case 5.—W. C., aged 23 at time of uroselectan investigations, Sept. 11, 1931.

HISTORY.—Congenital epispadias with incontinence. Previously several plastic operations performed without success. Left ureter transplanted in February, 1930. Right ureter transplanted in May, 1931. Post-operative notes state that on July 4, 1931, his general condition had much improved and that he had acquired satisfactory control of the rectum. A further note made on Aug. 6 is to the effect that his bowels move on an average four times daily and that he usually passes the night without evacuation being necessary.

UROSELECTAN FILMS (*Figs.* 408–411).—

Fig. 408 : Right—faint blurred shadow in renal area suggestive of hydronephrosis.
Left—faint kidney shadow showing clubbing and a moderate degree of dilatation of the calices. The ureter is faintly outlined above the ilium, is somewhat tortuous, and definitely dilated.

Fig. 409 : Right—kidney shadow increased in density and showing definite hydronephrosis. About an inch of the upper end of the ureter is shown and is dilated.
Left—renal and ureteric shadows less dense.
In the pelvis there is a shadow in front and to the left of the sacrum.

Fig. 410 : In both renal areas the shadows have become less dense and the outlines are indistinct.
Left—the outline of the dilated ureter is again seen faintly in that part of its course above the ilium.
The shadow in the pelvis has increased in size and density, extending upwards in front of the sacrum.

Fig. 411 : Both kidney and ureteric shadows have disappeared The shadow in the pelvis shows a slight increase in size and the large gut is outlined from iliac colon to cæcum by a shadow of varying density.

The blood-urea was 43 mgrm. per 100 c.c.

SUMMARY OF CONDITION OF KIDNEYS (4 months after transplantation of ureters).—Both kidneys show a definite degree of hydronephrosis, more marked on the right than the left, and the ureters are correspondingly dilated. While the faint shadows on the first film indicate some impairment of renal function, yet the shadow in the pelvis on the third film shows that quite a good excretion of the dye had taken place at the end of one and a half hours after injection.

LATEST REPORT (Aug. 5, 1935).—This patient is now in an institution because of his deteriorated mental condition. His general physical condition is fair. He has to empty the large bowel every two to three hours both day and night. Incontinence has occurred but on very rare occasions only.

FIG. 412.

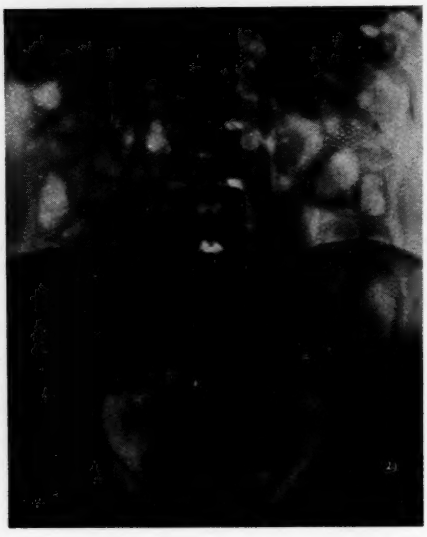

FIG. 413.

FIG. 414.

FIG. 415.

FIGS. 412–415.—*Case* 6. Uroselectan films taken 5 minutes, ½ hour, 1½ hours, and 6 hours respectively after injection of the dye.

Case 6.—Mrs. M. F., aged 35 at time of uroselectan investigation, March 21, 1932. When admitted for this purpose the patient was in excellent health and able to do her daily work.

HISTORY.—In 1926 the patient gave birth to a child after a very prolonged and difficult labour necessitating a forceps delivery. She noticed incontinence of urine immediately afterwards. The cause of this was found to be a vesico-vaginal fistula and up to 1930 several operations had been performed for the condition but without success. At this time the opening into the bladder easily admitted a finger. Transplantation of the ureters was advised as the only hope of terminating the miserable condition. The right ureter was transplanted in August, 1930, and the left one a month later. The patient made a good recovery from both operations.

UROSELECTAN FILMS (*Figs.* 412–415).—

Fig. 412: Right—shadow of good density showing normal calices, pelvis of kidney, and the ureter as far down as the transverse process of the last lumbar vertebra.
Left—kidney shadow of good density showing dilatation of both calices and pelvis; the ureter is outlined as far down as the upper border of the sacrum by a shadow of varying density and is shown to be considerably dilated, especially in its lower part.
Already a faint shadow has appeared in front of the sacrum.

Fig. 413: Right—kidney and ureteric shadow not quite so dense as in first film.
Left—shadow of both kidney and ureter of increased density; the lower part of the ureter is dilated in a bulbous manner.
The shadow in front of the sacrum is much denser than in the first film.

Fig. 414: Right—shadow of kidney and ureter now very faint.
Left—density of kidney shadow increased, that of ureteric shadow diminished. Below the kidney shadow is another, circular in outline—apparently bowel content.
The shadow in front of the sacrum has increased in size and density.

Fig. 415: Both kidney and ureteric shadows have disappeared. The rectum and lower pelvic colon are outlined by the dense shadow in the pelvis. The dye has reached as high as the splenic flexure of the colon, clearly outlining the iliac and descending portions.

The blood-urea was 44 mgrm. per 100 c.c.

SUMMARY OF CONDITION OF KIDNEYS (18 months after transplantation of ureters).—On the right side, hydronephrosis and hydro-ureter are present in marked degree. In spite of the obstruction which is causing this degree of dilatation, the good density of the shadow in the first film shows that the function of the kidney is good. The left kidney possesses normal structure and function.

In spite of several inquiries made in April and May, 1935, we have been unable to trace this patient again.

FIG. 416.—*Case* 1. MRS. E. S., aged 41, with her family, all born after transplantation of both ureters into the bowel nineteen years previously.

FIG. 417.—*Case* 2. Miss V. K., aged 24. In good health 18 years after double transplantation of the ureters.

SUMMARY

The results of this investigation of the pathological changes in the kidneys and ureters and the renal function in patients in whom the ureters had been transplanted into the colon are grouped together to form the table below in order that the reader may see them at a glance.

TABLE SHOWING PATHOLOGICAL CHANGES IN KIDNEYS AND URETERS TOGETHER WITH THE RENAL FUNCTION IN 6 CASES OF TRANSPLANTATION OF THE URETERS

Case No.	Right			Left		
	Kidney	Ureter	Renal Function	Kidney	Ureter	Renal Function
1	Hydronephrosis	Marked dilatation	Impaired	Normal	Slight dilatation	Good
2	Hydronephrosis Calculi	Not shown	Impaired	Normal	Slight dilatation	Good
3	Hydronephrosis	Marked dilatation	Impaired	Clubbing of calices	Not shown	Good
4	Normal	Slight dilatation	Good	Normal	Slight dilatation	Good
5	Hydronephrosis	Marked dilatation	Impaired	Hydronephrosis	Marked dilatation	Impaired
6	Hydronephrosis	Marked dilatation	Good	Normal	Normal	Good

Pathological Changes in the Kidneys and Ureters.—The outstanding change in these organs, as demonstrated by the uroselectan pyelography, is dilatation. In only one case (*Case* 6) did a kidney and its ureter appear perfectly normal, but four other kidneys, in *Cases* 1, 2, and 4, also showed a normal structure although the corresponding ureters were slightly dilated. The remaining seven kidneys were the seat of hydronephrosis of varying degree and the corresponding ureters in five of these were much dilated; no ureteric shadow was seen in the other two. It will be noted that in the first three cases the density of the shadow of the right kidney increased progressively in the second and third films, taken ½ hour and 1½ hours respectively after injection, indicating that the uroselectan was being excreted into the lumen of the kidney at a quicker rate than it was escaping into the bowel and thus showing the presence of some obstruction of the ureter. However, in only one of these three cases (*Case* 1) was the obstruction sufficient to have prevented the escape of all the dye from the kidney into the bowel at the end of 6 hours after injection, for a faint shadow of the upper calices and ureter was to be seen in the film taken at this time.

It is known that dilatation of the upper urinary tract may be the result of either obstruction or infection or that both may play a part, but the regularity of the

dilatation shown in the uroselectan pyelograms of these cases points to obstruction as the major causative factor.

In every case the original transplantation had been carried out by imbedding the ureter in the wall of the bowel and without the use of indwelling ureter catheters. Whether the slight obstruction demonstrated by this investigation is caused by the imbedding having been rather too thoroughly carried out or is really cicatricial and the result of a mild infection at the site of the anastomosis it is not at present possible to say. Another explanation, or at least a contributory cause, may be kinking of the ureter where it enters the bowel.

Renal Function.—The uroselectan proved that every kidney was a functioning organ, although in some the function was impaired. In two cases (*Cases* 4 and 6) the shadows of both kidneys on the first films, taken 5 minutes after injection, were of sufficient density to indicate that the dye was being excreted in a normal manner and that therefore the renal function was good. A similar condition was seen in the left kidney in three other cases (*Cases* 1, 2, and 3). In only one case (*Case* 5) did the shadows reveal an impaired function of both kidneys, while impairment of the function of the right kidney only, was noted in *Cases* 1, 2, and 3.

The chief point of interest concerning the impairment of function which had taken place in some of these kidneys lies in the fact that in none could it be considered gross, for in none was there a complete failure to excrete the dye. Further proof of this is apparent in the third films taken at the end of 1½ hours after injection; in all of these a shadow of good density is present in front of the sacrum, indicating that much of the dye has already been excreted by the kidneys and has passed into the rectum and lower part of the pelvic colon.

Blood-urea.—The values of the blood-urea in these cases lie between 35 and 48 mgrm. per 100 c.c. They are of interest because in the presence of such definite obstructive changes one might reasonably have expected higher values. They are, moreover, in agreement with the renal functions as indicated by the uroselectan and also with the general good health of these patients.

Renal Infection.—While, as previously mentioned, it is probable that in every case of transplantation of the ureters an ascending infection of the kidney takes place in some degree, yet evidence obtained in this investigation indicates that in the cases under review it must exist in only a very mild form, for the dilatation shown by the pyelograms is obstructive in type and does not bear the irregular characteristics of that due to infection. Furthermore, it is unlikely that severe renal infection would be accompanied by such a small degree of impairment of renal function and such low blood-urea values.

The Rôle of the Large Bowel in the Storage of Urine.—It is interesting to trace, through the shadows produced on the films by the uroselectan, the part played by the large bowel in the storage of the urine. In every case, an appreciable amount of urine had collected in the rectum at the end of half an hour after the injection of the dye. In another hour the urine had filled the rectum, and its level had begun to ascend into the pelvic colon, occupying its lower portion. By the end

of the sixth hour the urine, in four cases, had found its way back as far as the cæcum, and in another had risen to the level of the splenic flexure.

These films present, in a pictorial manner, convincing proof that, in cases where the ureters have been transplanted into the colon, not only the rectum but the remaining portion of the large bowel can, and does, act as the reservoir for the urine, and they explain at once why these patients can allow such lengthy intervals to elapse between evacuations.

We are much indebted to Dr. Whately Davidson, Honorary Radiologist to the Royal Victoria Infirmary, Newcastle-upon-Tyne, for the interest he took in these cases and for the help he gave us on many occasions.

REFERENCE

[1] TURNER, G. GREY, " The Treatment of Congenital Defects of the Bladder and Urethra by Implantation of the Ureters into the Bowel: with a Record of 17 Personal Cases ", *Brit. Jour. Surg.*, 1929, xvii, 114.

A GRIDIRON ACCESS TO THE BILIARY APPARATUS

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THE very multiplicity of incisions for approach to the gall-bladder and bile-ducts suggests the relatively unsatisfactory character of all, or at least an absence of general applicability to biliary cases. The paramedian is adequate in thin subjects and has the advantage of extensibility without parietal mutilation (surely discounted nowadays when the abdomen is opened on much more precise indications than formerly); the outer rectus incisions need extensions and are liable to nerve damage—their suture in the usual gall-stone case is difficult, particularly so in the instance of the posterior sheath, where the stitches cut out of the fatty atrophied tissue along the line of the fibres on the slightest tension; the paracostal incision of Kocher is nerve-murderous and bloody; the transverse incision of Maylard is sound with regard to the relationship between tension and the line of incision (wherein the vertical incisions are great offenders), but the division of the rectus is troublesone and bloody.

The approach here suggested is claimed to be:—

1. Preservative of parietal innervation.

2. Particularly easy of suture even with a straining patient, the stitches having no tendency to cut out.

3. Adequate in all cases, the access being equal to that in the Kocher method.

4. A gridiron access permitting the patient to get up very early without the slightest danger of bursting the wound.

5. Suitable for dealing with the appendix either through this incision or by a simple modification.

TECHNIQUE

1. The incision should reach the anterior sheath of the rectus $\frac{1}{2}$ to $\frac{3}{4}$ in. to the inner side of the outer edge of this muscle; where this is palpable the cut can be made accordingly, otherwise it passes downwards vertically from the region of the eighth costal cartilage for a distance appropriate to the habitus of the patient, varying from 5 to 8 in.

2. The anterior sheath is incised $\frac{1}{2}$ to $\frac{3}{4}$ in. inside the outer edge of the rectus; the margins of the outer flap being held by forceps; this is reflected outwards from the muscle, the inscription offering difficulty and several bleeding points having to be secured.

3. The crux of the access now is reached; the *posterior lamina of the internal oblique contribution to the rectus sheath is incised as it passes to the transversalis* (a muscle showing considerable variety in its formation of the upper part of the sheath), throughout the whole length of the incision (*Figs.* 418, 419). The oblique muscles are now found capable of wide retraction from the surface of the transversalis.

FIG. 418.—Shows the posterior lamina of the internal oblique aponeurosis perforated by an intercostal nerve. The line of section is indicated by an arrow.

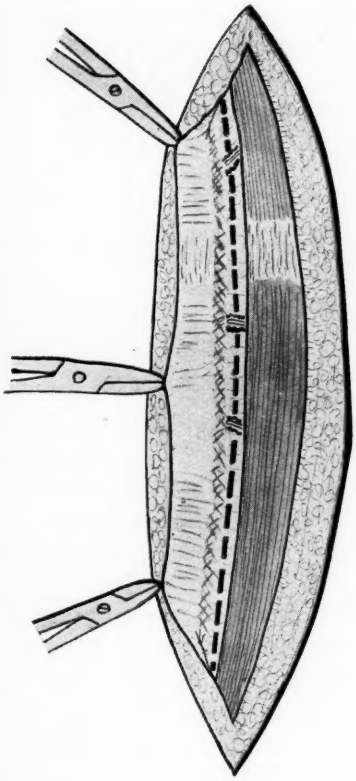

FIG. 419.

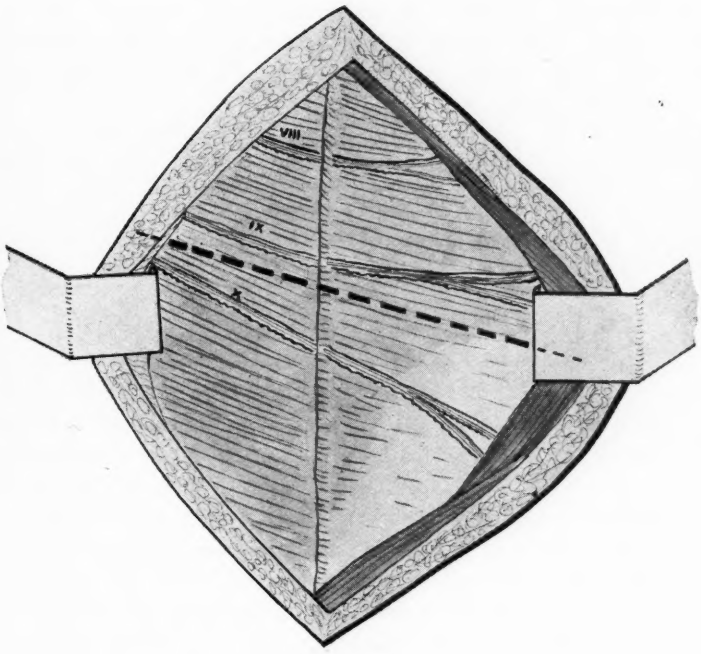

FIG. 420.

FIG. 419.—The outer flap of the rectus sheath drawn outwards, showing the splitting of the internal oblique aponeurosis (criss-cross line) and the position of incision in posterior lamina broken line).

FIG. 420.—Rectus and obliques retracted; the transversalis with the eighth, ninth, and tenth nerves and accompanying arteries and veins are seen. Incision of transversalis indicated by broken line.

4. The obliques and the rectus, freed by gauze, are widely retracted (the rectus, if very wide, may need some division from the seventh cartilage). The eighth, ninth, and tenth nerves are seen; the transversalis is divided almost transversely between the latter pair, which emerge close together from the costal margin; the division extends from the mid-line to the region of the tenth costal cartilage (*Fig.* 420).

FURTHER OBSERVATIONS

1. If the cæcum and appendix are not mobile, the latter may be dealt with by extending the division of the anterior sheath and posterior lamina to a lower level, and making an appropriate transverse incision in the posterior sheath.

2. Paramedian incision and *transverse division* of the posterior sheath gives adequate access and perfect safety from wound-bursting in cases of perforated duodenal ulcer.

3. The *posture of the patient* for biliary operations is ideally a sort of inverted Trendelenburg, but this is difficult of maintenance in the diffluent sort of patient usually met with in these cases; the essential is that gravity should be utilized to remove the intestine from the field of operation; hence the thorax and head of the patient may be on a level (supported by sand-bags and the bridge), the abdomen inclined downwards at an angle of 40° to 50°, and the lower limbs level. The bridge on the operating-table, if ever so slightly malposed, will cause the ducts to be drawn up into the thorax, and greatly intensify the difficulty of the operation.

MUCOID CARCINOMA OF THE CÆCUM IN A BOY OF 13 YEARS

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A REVIEW of the literature reveals a comparative dearth of reported cases of carcinoma of the colon in childhood. The following have been found :—

AUTHORS AND DATE	SITE IN COLON	AGE IN YEARS	SEX
Ball, H. A. (1935)	Sigmoid flexure*	15	F
Chajutin, D. M. (1929)	Cæcum	14	F
Clar	—	3	M
Desaive, P. (1934)	Anorectal	13	—
Garrard, W. A. (1897)	Sigmoid flexure*	12	M
Kanthack, A. A., and Furnivall, P. (1896)	Ascending colon*	17	M
Lawson, T. C. (1934)	Rectum	17	M
Nothnagel, H. (1898)	Cæcum	12	M
Pouzet, F. (1933)	Ascending colon	14	M
Raiford, T. S., and Buttles, E. M. (1933)	Rectum*	13	F
Rocher, H. L., and Guerin, R. (1931) ..	Splenic flexure	11	—
Schöning (2 cases)	Rectum (both)	17 (both)	F (both)
Wakeley, C. P. G. (1933)	Ascending colon	16	M
Walker, R., and Daly, J. F. (1934) ..	Cæcum	5	M

* Showed mucoid degeneration.

It is apparent that carcinoma of the cæcum in childhood is a rarity, and hence the present case seems worthy of record. After this case was described to the Edinburgh Pathological Club my attention was directed to a further instance of carcinoma of the (transverse) colon in a 15-year-old boy who was admitted under the care of Mr. Stuart in the Royal Infirmary, Edinburgh, in 1935.

HISTORY.—The patient was a schoolboy aged 13 years. Five days before admission he complained of severe pain in the right iliac fossa, the onset of which was shortly followed by vomiting. On the following day he was comparatively well, but three days before admission the pain and vomiting recurred and continued intermittently until he was admitted under the care of Mr. Struthers in the Royal Infirmary, Edinburgh. On the day of admission (Dec. 31, 1934) the patient's bowels moved twice. There was no history of any bladder trouble.

ON ADMISSION.—The boy's temperature was 99·2° F., his pulse was 80 per minute, and his respirations 22 per minute. His general condition was poor and he was obviously anæmic. His tongue was furred, but moist. His abdomen moved freely on respiration and showed neither distension nor peristalsis, but a swelling was visible in the right iliac fossa. On palpation this was found to be firm and tender and guarded by resistant muscles.

OPERATION.—On Jan. 1, 1935, the abdomen was opened through a gridiron incision in the right iliac fossa. A smooth, firm swelling was found in association with the cæcum, and some enlarged glands were found in the mesentery. On the basis of these findings a diagnosis of hypertrophic tuberculosis of the cæcum was made and an anastomosis was carried out between the lower part of the ileum and the middle of the transverse colon.

SUBSEQUENT HISTORY.—Convalescence was uneventful. The swelling in the right iliac fossa did not diminish, but became less tender and the patient's general condition improved greatly. He was sent to convalesce at the Astley Ainslie Institution on Feb. 12.

From the institute the patient was returned to the infirmary on April 10 with a history of having on that date had a recurrence of abdominal pain and vomiting. Following admission he was sick several times, but an enema produced a good result, and except for slight abdominal pain there were no further complaints. During the night and day following admission he had about fifteen fits, which appeared to begin on the left side and spread over the body. The contractions, at first clonic in character, became tonic, and were accompanied by partial loss of consciousness. After the last fit at 9.10 p.m. on April 11 he became drowsy, and though Kernig's sign remained negative definite neck rigidity was detected.

Thereafter the patient had no more fits, and after three days the drowsiness and neck rigidity disappeared. A small lump developed in the left occipital region, but apart from occasional headaches he remained well and was discharged again to the Astley Ainslie Institution on April 30. Whilst there his condition deteriorated; he lost weight, complained of spinal pain, became drowsy, and on May 10 he took three fits characterized by twitching of the right side of the face and of the eyes to the right side and by partial loss of consciousness. On lumbar puncture the cerebrospinal fluid was found to be under pressure and slightly turbid, but no tubercle bacilli were found.

He was re-admitted to the infirmary on May 14. The lump at the back of the head had enlarged and fluctuation could be elicited in it, but it was not tender. The scalp was movable over it, yet the lump itself appeared firmly fixed to the underlying bone. From the swelling a chain of enlarged glands extended down into the neck. He complained of pain in the neck, and though no actual neck rigidity was present he resisted attempts to lift his head. There was tenderness over the last cervical and first thoracic vertebræ. Kernig's sign was positive on both sides, but the pupillary, knee, ankle, and plantar responses were normal. Physical examination revealed nothing abnormal in the chest. X-rays revealed an area of lessened density in the occipital bone, nothing abnormal in the vertebral column, and diffuse mottling throughout the right lung, most marked at the hilum.

A needle was inserted into the occipital lump, but nothing could be aspirated. The swelling steadily increased. Twice he passed stools containing bright red blood, and continually complained of severe abdominal pain which necessitated heroin. On May 17 he had rigors and his temperature rose to 103°. Thereafter his temperature fell, but remained irregular. On May 21 he had a fit. His general condition deteriorated rapidly, he sweated profusely, became emaciated, and died on May 22. Clinically death was attributed to tuberculous meningitis.

POST-MORTEM EXAMINATION.—

Macroscopical.—The body was that of a rather emaciated boy. The abdomen was somewhat prominent, and a long, oblique, well-healed scar was present in the right iliac fossa. On opening the body the peritoneal cavity was found to be congested, especially in the lower quadrants, where there was some purulent exudate. Over the liver and diaphragm a number of small yellow nodules was seen in the serous membrane. The ileocolic anastomosis was apparently healthy.

The pleural sacs were partly obliterated by fibrous adhesions, and a small quantity of serous fluid was present in each.

The cæcum was hard to the feel, but not enlarged. On section its wall was found to be replaced by a dense yellowish-white mass which completely surrounded the narrow lumen (*Fig.* 421). The infiltration also involved the ileocæcal valve and

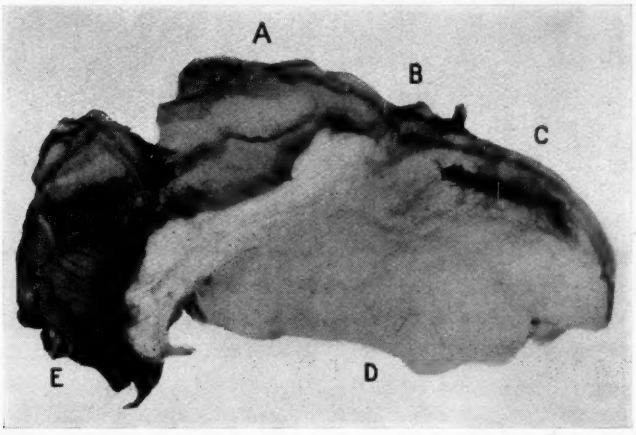

FIG. 421.—A, Cæcum with walls much thickened by malignant growth which has spread proximally through the ileocæcal valve (B) to involve the terminal ileum (C); D, A mass of lymph-glands replaced by neoplasm; E, Ascending colon showing precipitous margin of cæcal growth at proximal end.

the terminal 1½ in. of the ileum, where it gradually faded away. The distal margin of the cæcal mass, on the other hand, was precipitous and well defined. The inner surface of the cæcum was ulcerated and hæmorrhagic. In the ascending colon just distal to the neoplasm two or three small nodules suggesting secondary implants were found in the mucosa. The appendix, though swollen, did not appear to be involved.

The glands in the ileocæcal angle were enlarged and on section were found largely replaced by tumour tissue in which areas of necrosis were present. Lymph-node involvement was also found in the para-aortic, omental, and bronchial glands.

The spleen was enlarged and soft. The stomach, liver, gall-bladder, pancreas, kidneys, ureters, and bladder showed nothing of note. The heart was dilated and the myocardium pale and soft. Throughout the lungs were scattered a few small firm nodules of tumour tissue.

On removing the skull there was no evidence of meningitis, but small nodules of tumour were present in both anterior and posterior fossæ and in the right middle fossa. The largest skull-deposit measured 4½ cm. in diameter and was found in the left occipital region. It had grown so as to produce fairly large masses of gelatinous

tissue both external to the dura (*Fig.* 422) and under the scalp. The mass in the latter situation represented the swelling which had been aspirated during life. Apart from considerable œdema, the brain showed nothing of note.

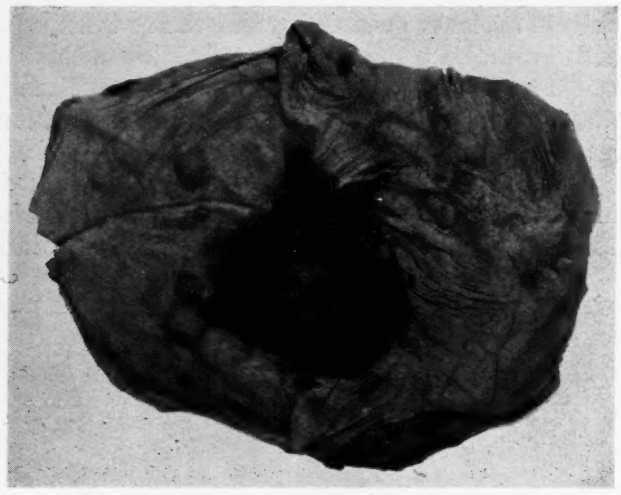

FIG. 422.—Dura from left occipital region with mass of gelatinous growth attached to its external surface. In the immediate neighbourhood of the mass are several small subsidiary nodules.

Microscopical.—The wall of the cæcum from mucosa to peritoneum was infiltrated by a spheroidal-celled carcinoma. Many of the cells showed the 'signet-ring' appearance of mucoid degeneration, and occasionally small masses of mucus

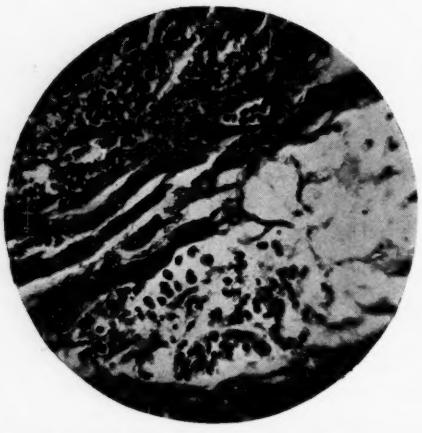

FIG. 423.—Cæcum showing infiltration of its muscular coat by spheroidal-celled carcinoma. Some of the cells have the 'signet-ring' appearance of mucoid degeneration, and to the right is a mass of mucus almost devoid of cells. (× 120.)

FIG. 424.—Lung showing perivascular lymphatic spread of the carcinoma. To the right of the vessel is a mass of spheroidal-celled growth in a state of advanced mucoid degeneration: to the left is œdematous lung tissue. (× 50.)

devoid of cells were present between the muscle-bundles (*Fig.* 423). The surface of the growth was necrotic, ulcerated, and much congested.

A bronchial gland showed almost complete replacement of its substance by spheroidal-celled neoplasm undergoing early mucoid change.

Sections of lung demonstrated a perivascular lymphatic spread of the growth (*Fig.* 424). The latter was spheroidal-celled and in a state of advanced mucoid degeneration. From the lymphatics the neoplastic tissue had spread by direct infiltration into the adjacent lung tissue so that nodules of considerable size had been formed. The wall of a fairly large vein was seen being invaded by tumour-cells (*Fig.* 425).

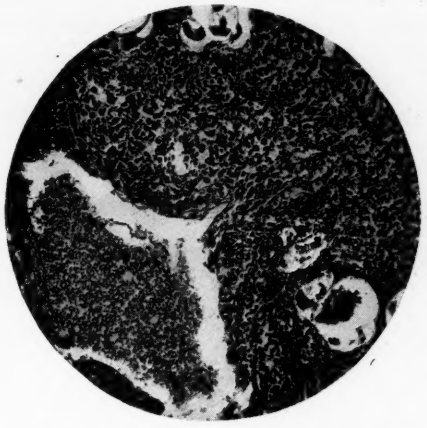

FIG. 425.—Lung showing perivascular lymphatic spread of the carcinoma. The spheroidal-celled mass to the right has invaded the wall of the adjacent vein. (× 50.)

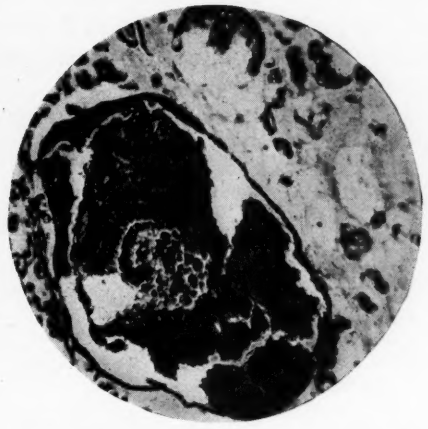

FIG. 426.—Scalp nodule in left occipital region showing a large thin-walled vessel surrounded by mucoid growth and containing a malignant embolus in its lumen. (× 120.)

The scalp nodule consisted of spheroidal-celled tissue in a state of advanced mucoid degeneration. Two or three thin-walled blood-vessels showed small masses of tumour-cells within them (*Fig.* 426).

Liver showed fatty change but no secondary deposits.

COMMENTARY

The outstanding feature of the case is the age of the subject. During the ten years 1920–9 inclusive 322 cases of carcinoma of the colon, excluding rectum, were admitted to King's College Hospital and the average age of the patients was 52 (Wakeley, 1933). In 100 cases of carcinoma of the cæcum collected from the Mayo Clinic by Craig and MacCarty (1923) the average age incidence was 49, while in 25 cases of cæcal carcinoma operated on by Wakeley (Wakeley and Rutherford, 1932) the average age incidence was 58. No doubt the rarity of the condition at the age of 13 years was largely responsible for the way in which the surgeon was misled in making a diagnosis of hypertrophic tuberculosis of the cæcum, and it is interesting that the provisional clinical diagnosis in Ball's (1935) case of carcinoma of the sigmoid flexure in a girl of 15 was tuberculous peritonitis.

The mode of spread throughout the greater part of the illness appears to have been lymphatic—first to the regional mesenteric glands and thence by the para-aortic to the glands of the lesser omentum and to the bronchial glands, whence there was perivascular spread into the lungs. A pulmonary vein in process of being eroded was observed microscopically and it is likely that it was by such a process that invasion of the blood-stream occurred and metastases in the skull ensued. The

absence of secondary growths in the liver is noteworthy, but by no means exceptional in cases of cancer of the colon. This case is thus similar to that of Raiford and Buttles (1933), which was also characterized by skull metastases but no liver involvement.

Mucoid degeneration was an outstanding feature of the growths in this case, particularly of that in the occipital region. Such a change is a fairly common characteristic of colon carcinomata arising in the later years of life, and it would appear to be equally common in the earlier years. Thus of the 17 cases mentioned in this paper 5 exhibited the phenomenon.

I am indebted to Mr. Struthers for permission to publish this case.

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ANTERIOR DISLOCATION OF THE HIP

By J. A. MacFARLANE, Toronto

On the evening of Feb. 25, 1934, a schoolboy, aged 17, was admitted to the Toronto General Hospital complaining of severe pain in the left groin and inability to walk. About three hours previously he had fallen while ski-ing. He was going down a partially wooded hill and his left ski caught in some underbrush while he was

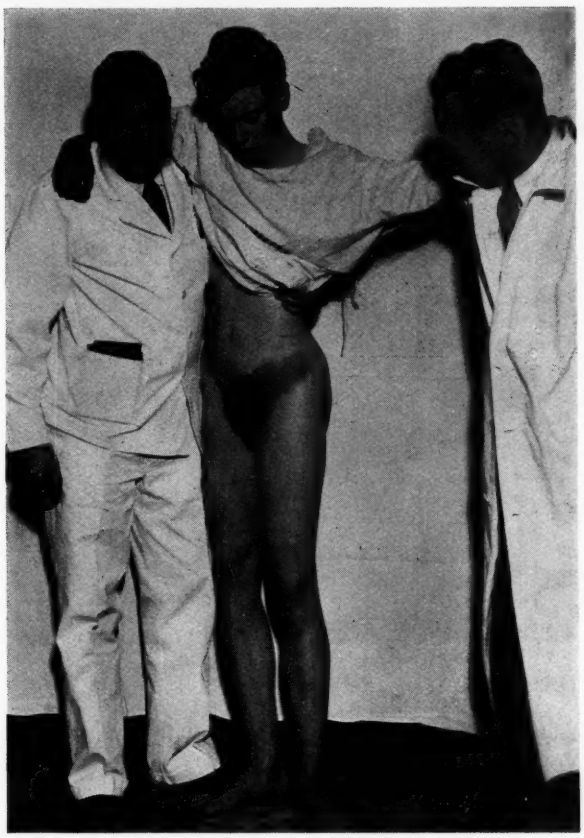

Fig. 427.—Anterior dislocation of the hip.

travelling at a good speed. He found himself lying prone, with his head down the hill, his right ski free of his foot and his left one turned out. The accident happened so quickly that he could not describe it more fully. He found he was unable to walk and had pain in the region of the hip-joint.

The boy was seen in the X-ray Department of the hospital, lying on a stretcher. The left leg was externally rotated and it appeared slightly longer than the right.

Measurements revealed no appreciable difference in the length of the limbs. The left thigh was in complete extension. There was no apparent adduction or abduction. There was a rounded prominence evident below Poupart's ligament. Active and passive movements at the hip-joint were absent. A diagnosis of anterior dislocation was made and X-ray plates confirmed this diagnosis. (*Figs.* 427, 428.)

The patient was placed on a mattress on the floor, given ether anæsthesia forthwith, and an attempt was made to reduce the dislocation. All attempts at reduction failed, and after thirty minutes of effort the patient was sent to the ward.

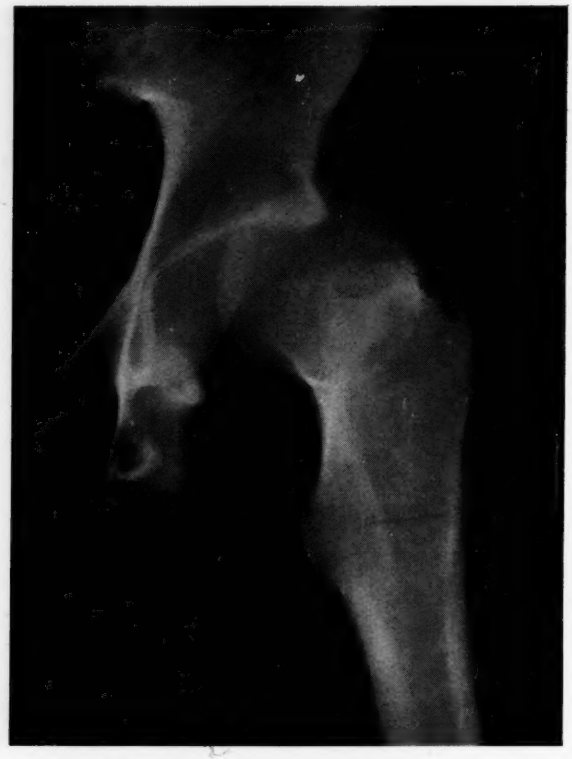

FIG. 428.—X-ray appearance of anterior dislocation of the hip.

On the following morning the physical findings were unchanged except for the presence of considerable bruising on the left groin. The boy was then anæsthetized again with full ether anæsthesia, and in the presence of several of the surgical staff further attempts at reduction were undertaken. The manipulations carried out were those described by Bigelow for anterior dislocations. The thigh was first flexed and pulled down, counter-extension being applied by a folded sheet around the opposite thigh and held by an assistant. With the thigh flexed and pulled down, pressure was applied over the prominence of the head, attempting to push it downwards and backwards as the thigh was rotated. These manipulations were carried out repeatedly by several surgeons in turn. At the end of forty-five minutes it was decided that further attempts were useless and might cause additional harm.

Two days later an open operation was carried out through a Smith-Petersen incision. The structures arising from the anterior part of the iliac crest were removed subperiosteally and retracted laterally, the sartorious retracted medially. This procedure exposed the head of the femur partially covered by some torn fibres of the rectus femoris. When this structure had been retracted laterally one tried to discover whether any muscular structure had slipped behind the head preventing its reduction. The psoas tendon was in its proper position and the dislocation seemed to be through the capsule between the medial border of the Y ligament and the pubocapsular ligament. Manipulations at this stage failed absolutely to dislodge the head from its position on the pubic ramus. The neck of the femur was further exposed by cutting the capsule towards its femoral attachment, and, in so doing, part, at any rate, of the medial limb of the Y ligament was cut. It was then possible to pass a long skid down into the acetabulum in front of the head and by combined efforts of flexion and pressure downwards on the head, guided as it was on the skid, and levered away from the pubic ramus, reduction was finally effected. The rent in the capsule appeared as indicated in *Fig.* 429.

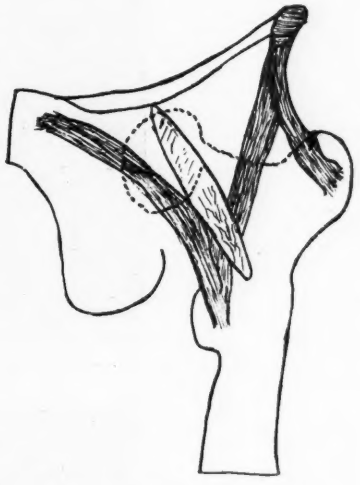

FIG. 429.—Appearance of hip after reduction.

The boy's convalescence was uneventful and he was discharged from the hospital in four weeks, walking with crutches. At the end of seven weeks he was allowed to walk without support. On Nov. 28 he was examined again. There was no limitation of motion in any direction and he was satisfied he could do anything he did previous to the accident. X-ray examination at this time showed no evidence of change in the femoral head. Examination in January, 1935, confirmed the findings of November, 1934 (*Fig.* 430). X-ray examination, however, at this time showed a little flattening of the head in the area of attachment of the ligamentum teres (*Fig.* 431).

COMMENT

Traumatic dislocation is a relatively rare injury. The dislocated head may go forwards or backwards. Posteriorly the head may be above or below the tendon of the obturator internus. Anteriorly it may be in the obturator foramen, it may rest on the pubic ramus, or it may be just below the anterior superior spine. I have personally treated only three cases of traumatic dislocation, two posterior ones and the case reported—a suprapubic one. In the files of the Toronto General Hospital there are only 26 cases of traumatic dislocation and these were all anterior.

Dr. R. M. Wansbrough has kindly allowed me to look into the records of the Children's Hospital for the past ten years. During that time there were four cases of posterior and one of anterior dislocation. Choyce[1] reported 59 cases of traumatic dislocation in children, this being the entire number in the literature reported up to that time. Three were suprapubic, one of which was reduced

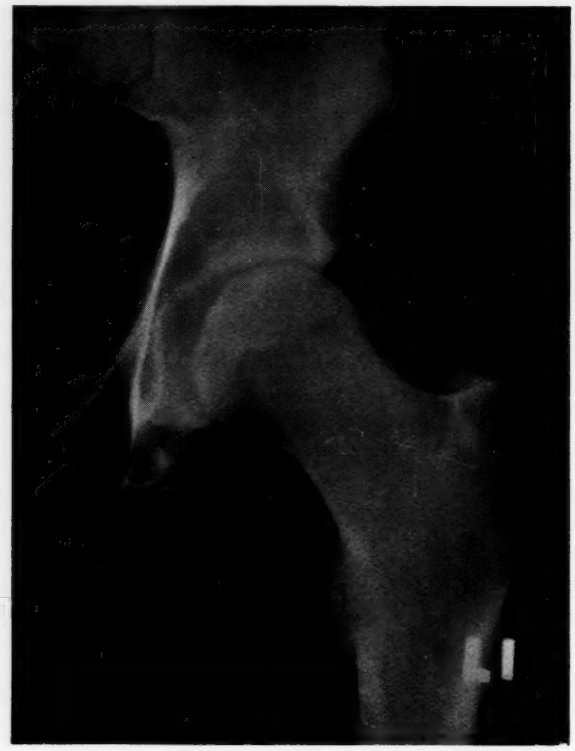

FIG. 430.—X-ray appearance after reduction (November, 1934).

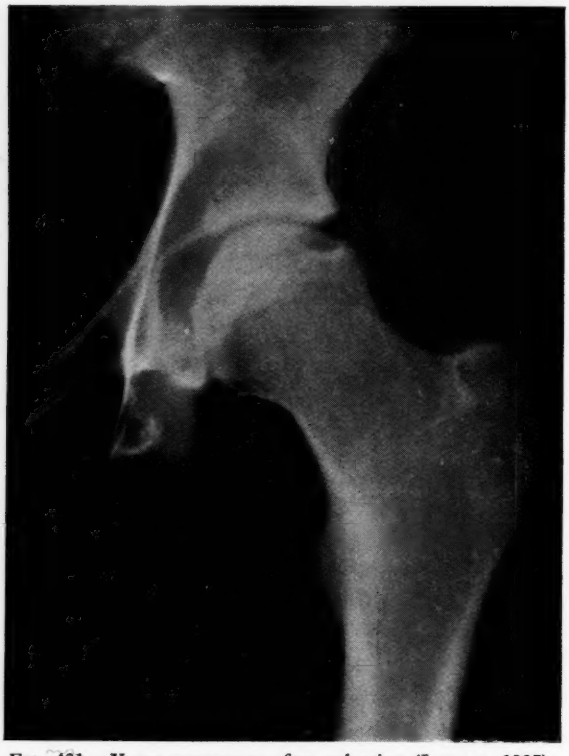

FIG. 431.—X-ray appearance after reduction (January, 1935).

immediately, and two of which necessitated open operation two and a half and three months following the injury. E. J. Smith[2] reported a case of suprapubic dislocation treated successfully by manipulation seven hours after the injury.

There are several reports in the literature of the treatment of late cases of traumatic dislocation, but I have not been able to find a case record which notes the difficulties which may be encountered in immediate reduction of suprapubic dislocations. Bigelow, in his monograph on dislocation of the hip states, " I have never met with suprapubic dislocation in the living subject and am therefore unable to speak of the extent of a difficulty of flexion alluded to by some writers as characteristic of this luxation." His experimental work on the cadaver led him to believe that this dislocation like all others might successfully be treated by manipulation.

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CALCIFIED CYST OF THE PERICARDIUM

By A. DICKSON WRIGHT, London

Calcified cysts of the pericardium are of sufficient rarity to warrant presentation of the following case.

A young man, a rubber planter, aged 25, had received a heavy blow on the chest while playing Rugby football in the tropics. Persistent pain after the injury resulted in an X-ray being taken, and the extraordinary calcified shadow shown in *Fig.* 432 was discovered. The patient was shown the X-ray and from then on

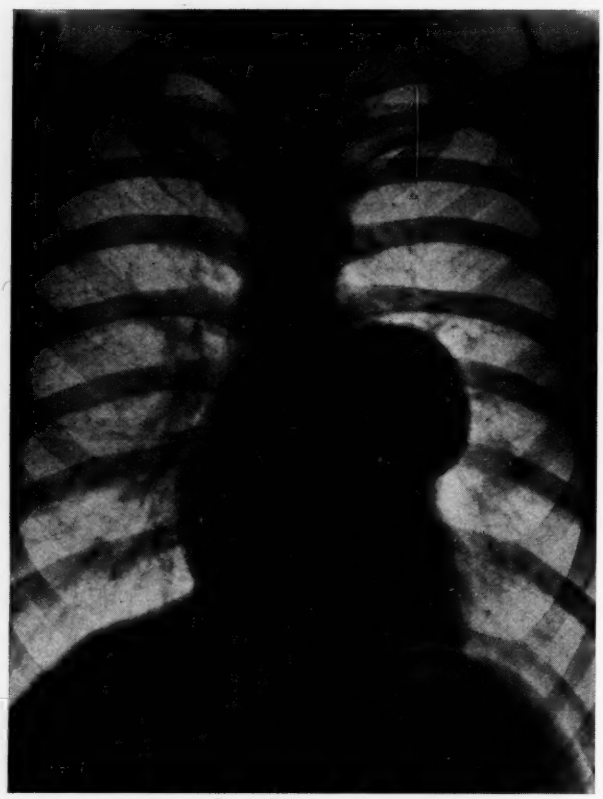

Fig. 432.—Antero-posterior view to show the position of the tumour. The lateral view showed it to be lying in contact with the anterior chest wall.

developed fluttering and disagreeable sensation in the region of the heart; he was referred to me with a view to having the tumour removed. There was one significant fact in his history in that seven years previously while playing hockey he had received such a severe blow with the ball over the heart that he became unconscious for some hours and was in bed for a week, taking two months to get quite well.

On the X-ray screen the tumour gave a most remarkable appearance of moving with the heart-beats. Artificial pneumothorax was induced to see if the tumour

moved with the collapsed lung, but as it did not do so it was inferred that it was attached to the pericardium. It was interesting to note that the pneumothorax greatly increased the fluttering sensations in the chest, probably because of the increased mobility of the tumour no longer surrounded by lung, and this was borne out on screening the patient, when the movements of the tumour were much accentuated.

OPERATION.—A transverse incision was made in the 5th intercostal space anteriorly, and the 5th and 6th costal cartilages and the 6th rib in the anterior axillary line were divided to increase the spread obtained with a Nelson rib retractor. The internal mammary artery was doubly ligated and the pleural cavity opened widely, and the tumour presented a most remarkable sight as it danced wildly about with the ventricular pulsation. The tumour was steadied with Allis forceps and

FIG. 433.—The cyst sawn through. The facet which contacted with the chest wall can be seen on the left half of the cyst.

an incision made round its base, the tumour being shelled out from its capsule. Two arteries required ligature in the bed of the tumour and it was then noticed that the pericardial cavity had not been opened, so that the tumour lay between the layers of the pericardium. The mediastinal pleura was sewn up with a continuous fine catgut stitch and the pleura was closed with pericostal sutures. A stab-drain was left through a rib space posteriorly, a mushroom-headed catheter being used for this purpose. This drain was water-sealed and yielded 10 oz. of blood-stained fluid in the first twenty-four hours ; the drainage then ceasing, the tube was removed after forty-eight hours.

Recovery was uneventful, and the patient has remained fit and well ever since and again plays Rugby football regularly within a few miles of the equator.

PATHOLOGY.—The tumour (*Fig.* 433) was strong and hard and had a facet on

one surface where it was presumed it rubbed against the chest wall. To open it a hack saw was used; it had a wall $\frac{1}{4}$ in. thick and contained a loculated brown mass of amorphous material with a beige-coloured fluid in the interstices, which fluid glistened with cholesterin crystals. The chemical examination of the fluid made by Dr. Roche Lynch showed the presence of an appreciable amount of iron; under the microscope crystals of cholesterin and calcium phosphate were numerous; no hooklets, hairs, or keratin were found.

Dr. W. B. Newcombe examined the structure of the cyst wall (*Figs.* 434, 435) and found it to consist of laminated fibrous tissue impregnated with calcium salts, and it was his opinion that the tumour was a calcified blood cyst.

FIG. 434.—Low-power view of the wall of the cyst, showing the laminated arrangement of the fibrous tissue. There was no endothelial or epithelial lining to the cyst.

FIG. 435.—High-power view of the cyst wall, showing the breaking up of the collagen fibrils and calcareous particles not removed in the process of decalcification.

Comment.—This case is an unusual manifestation of the well-known tendency for calcification to occur in the pericardium. The sequence of events was that the blow with a hockey ball in 1926 caused a large extravasation of blood between the layers of the parietal pericardium. The amount of blood was more than the local histiocytes could absorb, with the result that a large more or less spherical collection of semi-clotted blood was left between the layers of the pericardium. The irritation produced by this caused an adventitious fibrous capsule to be formed, which, like other chronic pericardial lesions, became calcified, and the blood contained in the chalky capsule could not be absorbed and in the slow process of years changed into cholesterin and other substances, but retained still a brownish colour from the blood pigments.

A CHLORIDE-SECRETING PAPILLOMA IN THE GALL-BLADDER

A TUMOUR OF HETEROTOPIC INTESTINAL EPITHELIUM CONTAINING PANETH CELLS AND ENTEROCHROMAFFINE CELLS AND ASSOCIATED WITH MASSIVE CHLORIDE LOSS: WITH A CRITICAL REVIEW OF PAPILLOMA OF THE GALL-BLADDER

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THE case to be described is one of cholecystostomy for presumed mucocele in which, during a drainage period of thirty-four days, there was an excessive outpouring of chloride and water; the source of this fluid was revealed only on subsequent cholecystectomy, when there was found in the gall-bladder a papilloma composed of intestinal epithelium. It is presented in detail on account of the difficulties in diagnosis, the peculiar biochemical behaviour, and the unique underlying pathology.

CASE REPORT

HISTORY.—A labourer, aged 64, was admitted to the Western Infirmary, Glasgow, on Oct. 30, 1934, under the care of Mr. Roy Young. He complained of constipation and of a swelling in the abdomen. During the preceding ten weeks he had noticed increasing constipation though his bowels had previously been regular. He first became aware of a swelling in the upper right abdomen three weeks prior to admission, and this did not, to his knowledge, vary in size during the intervening period. His appetite had recently been poor and he had been much troubled by flatulence and sour mouthfuls. He was losing strength and weight. No symptoms referable to the urinary system were elicited. He had suffered from pneumonia twenty and ten years previously. The family history was good.

ON EXAMINATION.—The temperature and pulse-rate of the patient were normal. He did not look acutely ill but was frail and his appearance suggested that he had lost some weight. There was no jaundice.

The right side of the abdomen was rather prominent and a smooth firm mass was palpable extending from the costal margin above to just below the intercristal line, and from the right flank to the middle line. The mass was not tender, was dull to percussion, and moved fairly freely with respiration and on manipulation. It could not, however, be readily pushed forward from the posterior lumbar region. The liver dullness was found to be normal except where its continuity with the dullness of the mass prevented the definition of its lower margin. There was slight epigastric tenderness.

Varicose veins were noted in the left leg. Occasional crepitations were heard at the base of the left lung. The heart and nervous system presented no abnormalities. The urine was normal and the Wassermann reaction was negative.

A barium enema passed freely to the cæcum. The mass was reported to lie medial to the cæcum and ascending colon and below and behind the transverse colon. It was not attached to any portion of the colon.

Cystoscopy and right-sided pyelography failed to reveal any abnormality in the urinary tract.

DIAGNOSIS.—In view of the paucity of symptoms, the diagnosis rested largely on the objective findings. Despite the marked constipation the barium enema appeared to eliminate primary disease of the colon. The mass seemed to lie too far forward to have its origin in the right kidney and this was supported by the pyelographic findings. The upper end of the mass was thought to pass too deeply for a cyst or tumour of the liver, and, though mucocele of the gall-bladder was considered with some favour on clinical grounds, the observation that the mass lay behind the barium-filled transverse colon appeared to exclude this condition. The pre-operative diagnosis was thus of a cystic or solid tumour of the mesentery or possibly of a pancreatic cyst. The radiological observation, however, that the mass lay behind the colon was proved at operation to be fallacious.

FIRST OPERATION.—On Nov. 20, Mr. Roy Young operated under general anæsthesia. Through an upper right rectus incision, he found the mass to consist of an enormously

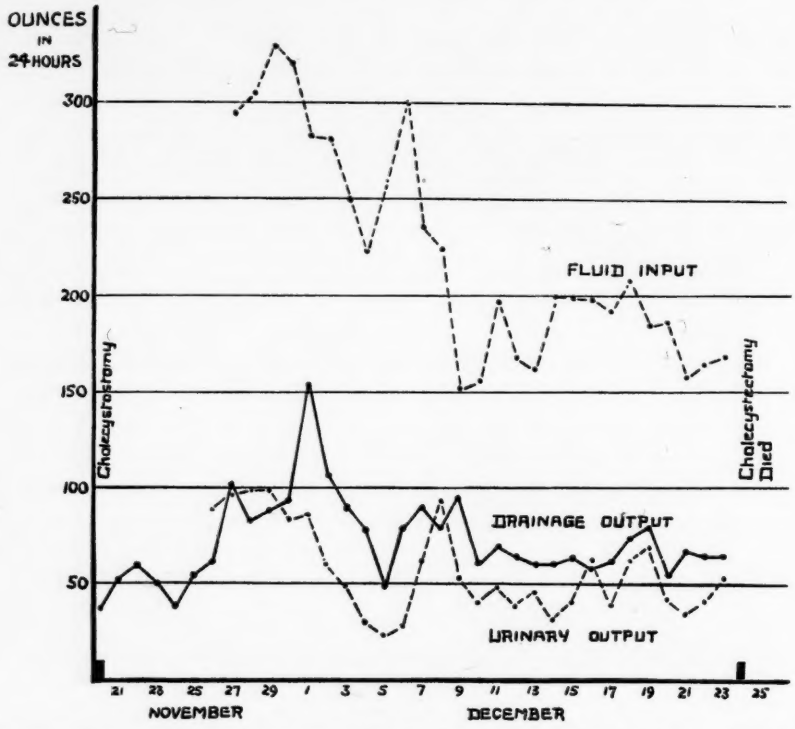


FIG. 436.—Chart showing drainage output from the gall-bladder, urinary ou put, and fluid input (including intravenous input) during the period of drainage.

distended gall-bladder occupying the usual site in front of the colon. Over 20 oz. of clear colourless fluid were aspirated. The gall-bladder was then incised and 10 oz. of thicker dirty fluid were removed along with eight rather flattened opalescent gall-stones. The patient's general condition was giving rise to some anxiety and drainage of the gall-bladder was therefore adopted in preference to its removal.

The fluid from the gall-bladder showed no organisms in films or on culture.

The centre of each calculus was found to consist of bile pigment admixed with a small proportion of calcium salts, while the pearly outer coating was of almost pure cholesterol. No unusual constituent was found.

POST-OPERATIVE COURSE.—During the twenty-four hours succeeding operation, 38 oz. of fluid, thin and at first somewhat blood-stained, drained from the gall-bladder. Thereafter the discharge continued as a clear and quite colourless watery fluid, and during the third day its amount reached 60 oz. in twenty-four hours (*Fig.* 436).

During this period the patient's general condition was satisfactory apart from cough and muco-purulent expectoration, but it gradually deteriorated, and by the fifth day (Nov. 25) he was obviously distressed. Cyanosis, pallor, and irregularity and weakness of the pulse indicated the onset of cardiac failure, while his pinched face, sunken eyes, and dry tongue pointed clearly to dehydration despite the fact that he had been drinking freely. The similarity of the clinical picture to that of intestinal obstruction led to an examination of the urine for chlorides. The urine contained practically no chlorides. In marked contrast to this was the finding in the drainage fluid of abundant chlorides. The intravenous administration of 2 pints of 2 per cent sodium chloride was followed within an hour by dramatic clinical improvement and the early re-appearance of chlorides in the urine. From this point saline was given by mouth in addition to water, and the total fluid input and urinary output were recorded (*Fig.* 436).

Quantitative estimations two days later (Nov. 27), gave the following results :—

	Urine	Drainage Fluid
NaCl ..	.. Trace	.. 0·79 per cent
Urea ..	.. 1·6 per cent ..	.. Not examined
$NaHCO_3$	.. Not examined	.. Negligible

Although chlorides were already practically absent from the urine, it was not until the following day that the patient's clinical condition again began to deteriorate, and by Nov. 30 he again showed all the adverse signs noted on Nov. 25. A similar intravenous injection of saline was therefore given and the clinical improvement was as striking as on the first occasion.

Until Dec. 24, a total period of thirty-four days, his condition was satisfactorily maintained and even improved by a series of such saline injections, each necessitated by the recurrence of the same group of symptoms at intervals of five or six days. The drainage amounts for this period, including the extraordinary peak figure of $7\frac{3}{4}$ pints on the eleventh day, are seen in *Fig.* 436.

The blood-urea was found on Dec. 7 to be 0·039 per cent.

Biochemical examinations immediately before intravenous injection on Dec. 11 gave the following results :—

	Urine	Drainage Fluid	Blood Plasma.
NaCl ..	.. Trace ..	.. 0·81 per cent	.. .. 0·52 per cent
Urea ..	.. 1·7 per cent ..	Not examined	.. .. Not examined

The drainage fluid was examined bacteriologically on Dec. 1. Abundant pus cells were found and a coliform organism was isolated and later identified as a paracolon bacillus.

Second Operation.—On Dec. 24, Mr. Roy Young again operated, on this occasion under spinal anæsthesia. The gall-bladder was freed from omental adhesions and removed without much difficulty. The patient's condition appeared satisfactory after the operation, but a few hours later he became very restless and noisy. His pulse weakened and he died eighteen hours after cholecystectomy. Permission for a post-mortem examination could not be obtained.

PATHOLOGICAL REPORT ON GALL-BLADDER

The gall-bladder (*Fig.* 437) forms an elongated sac, 16·5 cm. long by 10 cm. in maximum circumference, the fundus or distal end being somewhat narrower and showing the drainage opening. The external surface is in general smooth, but omental adhesions are noted at the fundus and also near the middle of the specimen, where there is present a circular constriction. The superficial veins are prominent, especially towards the broad end of the sac, where are also seen the cut end of the cystic duct and the cystic lymph gland. This part of the gall-bladder looks and feels almost solid owing to the tumour within. The sac wall, which has a red velvety lining and is in general firm to the touch, is about $\frac{1}{8}$ in. thick, except

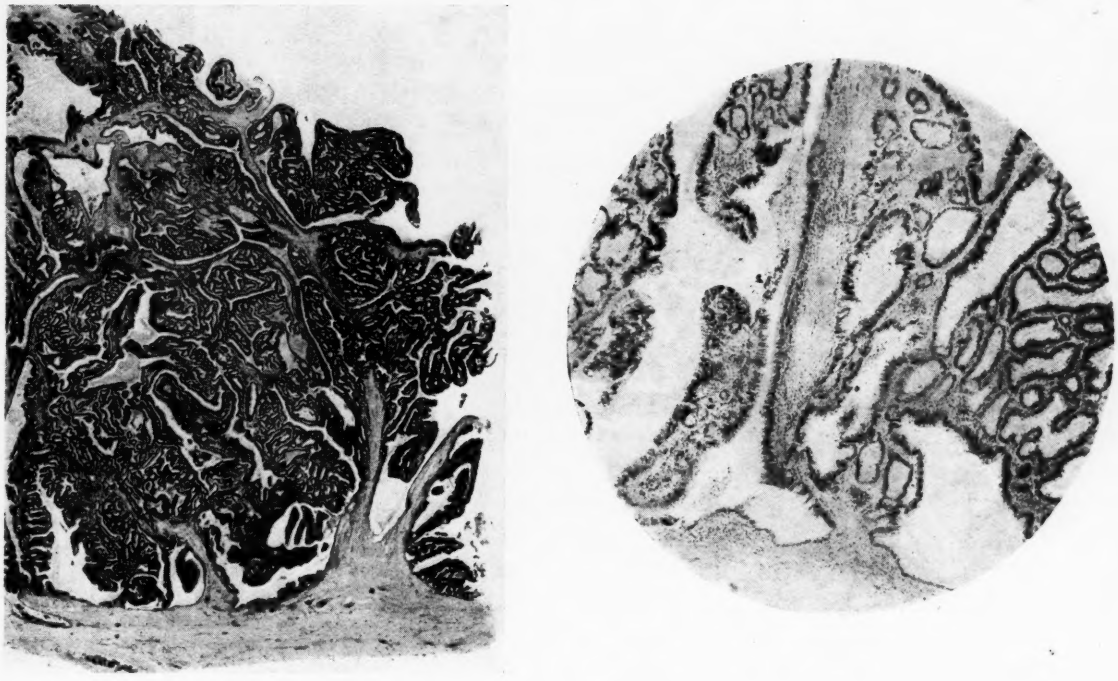
at the point of drainage, where there is an irregular dense thickening. Some 7·5 cm. from the orifice there is a thin projecting shelf corresponding to the constriction mentioned above; in part of its extent this is formed by all the components of the wall, the remainder being of mucosa alone.

On opening the region of the neck, a massive tumour is seen. This is a cauliflower-like growth, 3 cm. high, which is present as three main masses with numerous subsidiary and closely adjacent polyps; indeed, the proximal 5 cm. of the gall-bladder lining is almost entirely tumour-bearing. The main masses have a uniformly fleshy colour and appear almost solid; their surface, however, shows marked irregularity both gross and fine in type and on close examination they are seen to be essentially papilliform.

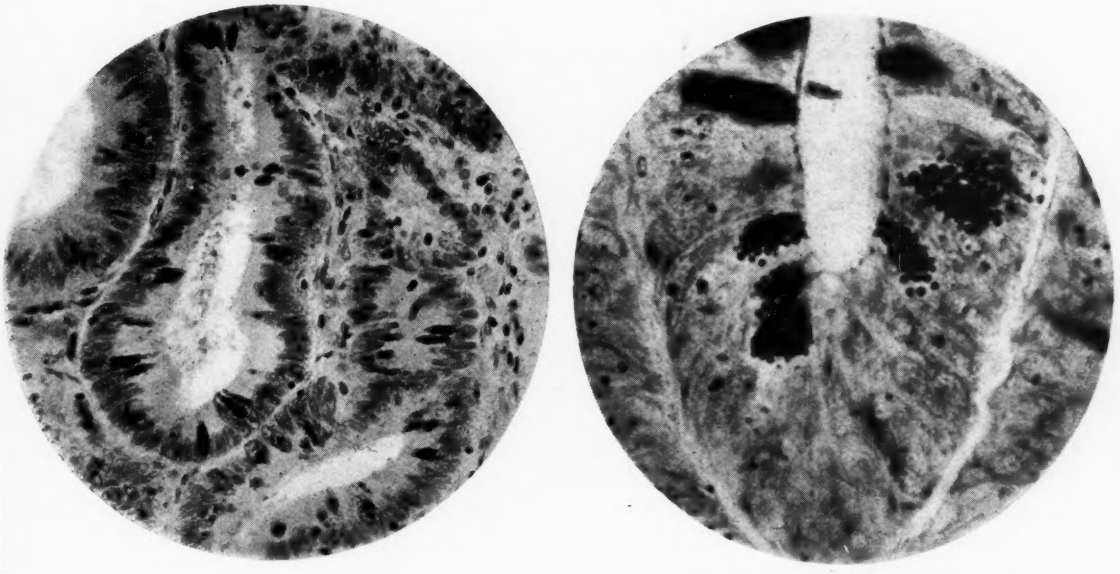
FIG. 437.—Inner wall of gall-bladder removed at second operation, showing at the upper end a large papilloma and at the lower end the thickened mass alongside the drainage opening. (× ½.)

Several portions of the general gall-bladder wall were examined histologically. In all of these the serous coat is ill defined; the muscular coat likewise is poorly demarcated and throughout its breadth the muscular fibres are much interrupted by œdematous fibrous tissue. The blood-vessels show œdema of their walls and, in places, slight endarteritis. There are also throughout the wall many small localized areas of infiltration of lymphocytes, plasma cells, and some endothelioid cells; in a few of the larger of these areas the cells are mainly polymorphs, with an unusual concentration of eosinophils at the margin. At the internal surface the inflammatory cells form an almost complete boundary zone; in parts this is surmounted by gall-bladder epithelium. The infiltrated layer is composed almost entirely of plasma cells; there are also a few lymphocytes and an occasional polymorph, this latter type being seen especially in the prominent congested young capillaries of this zone. The epithelium is of the usual columnar form with basal nuclei of oval rather vesicular type. Crypt formation is present, but is of shallow type, the crypts nowhere extending more than a short way into the muscle coat; they are completely lined with epithelium. Toward the neck there is less evidence of inflammation, but crypts are also present in this situation.

Section of the thickened mass at the point of drainage shows a chronic inflammatory tissue of dense fibrous type with hyaline change both in the stroma and in the walls of a thrombosed vessel; lying among this fibrous tissue is a small amount of blood pigment. Covering the mass there is an almost intact sheet of



FIGS. 438, 439.—Photomicrographs showing the complex villous structure of the papilloma. *Fig.* 438, hæmalum and eosin. (× 7.) *Fig.* 439, hæmalum and eosin. (× 35.)



FIGS. 440, 441.—Photomicrographs showing distribution and characters of the Paneth cells in the epithelium of the papilloma. *Fig.* 440, Gram's stain (modified) and carmalum. (× 250.) *Fig.* 441, phosphotungstic acid hæmatoxylin. (× 900.)

gall-bladder epithelium with slight crypt formation and an underlying zone of infiltration as described in the general sac wall.

The cystic duct shows a normal epithelium with mild chronic inflammation confined to the submucosa. In the cystic gland there is hyperplasia of the lymph follicles. The cystic artery shows hypertrophic nuclei in the muscle cells of the media and a slight fibrocellular intimal thickening.

The tumour itself presents the features of a complex papilloma (*Figs.* 438, 439). The stromal core is everywhere thin and shows only a few blood-vessels which, however, are markedly congested. Scattered irregularly throughout the stroma are areas of infiltration with inflammatory cells; these are mainly lymphocytes, but polymorphs are seen nearer the base of the tumour and also occupying an intra-epithelial position in some of the outer fringes of the papilloma. Despite the apparently compact character of the tumour, section of the marginal area shows the attachment to be in the form of numerous tendril-like stalks. No suggestion of malignant invasion of the wall by tumour is seen in any of the sections examined, but among the stalks there are a few crypts lined by an epithelium of the same type as in the papilloma.

The epithelium of the papilloma is in general tall and columnar in type. The elongated nuclei form an irregular crowded layer on the basement membrane; mitosis is occurring but is infrequent in proportion to the overcrowding of the cells. The cytoplasm of the cells is well stained and somewhat basophilic; at the free edge it shows a striated border which is considerably more marked than in the epithelium of the general gall-bladder wall.

In the hæmalum and eosin sections of the tumour, many of the epithelial cells are seen to contain refractile eosinophilic granules; these cells are easily distinguished under the usual high-power magnification of 360. The following staining reactions are noted for the granules:—

Reinke's neutral gentian	Purple
Bensley's orange G, acid fuchsin, and toluidin blue mixture	Fuchsin red
Heidenhain's iron hæmatoxylin	Black
Maximow's eosin-azur mixture	Red
Mann's eosin and methyl blue	Mainly red
Van Gieson's stain	Red
Malachite green and acridine red (*Fig.* 442)	Bright green
Mallory's phosphotungstic acid hæmatoxylin (*Fig.* 443)	Dark purple black
Gram's stain	Purple
Celestin blue, gallic acid eosin, and light green	Bright red
Celestin blue alone or with alcoholic chromotrope 2R	Unstained
Masson's silver method	Not impregnated

The granules are of various sizes; in some cells they are uniformly small and the cell stretches as a narrow band of tiny granules from basement membrane to free edge; in most, the inclusions are larger and the cells show a globular distal portion distended with granules; in others the granules are of different sizes; an occasional cell contains as few as four or five granules, but these are as large as 3 μ in diameter. Here and there the granules are seen in the process of extrusion and occasional clusters are apparent lying free.

The nucleus of the granular cell lies towards the basal part of the cell and shows no characteristic difference from the surrounding nuclei. None of the

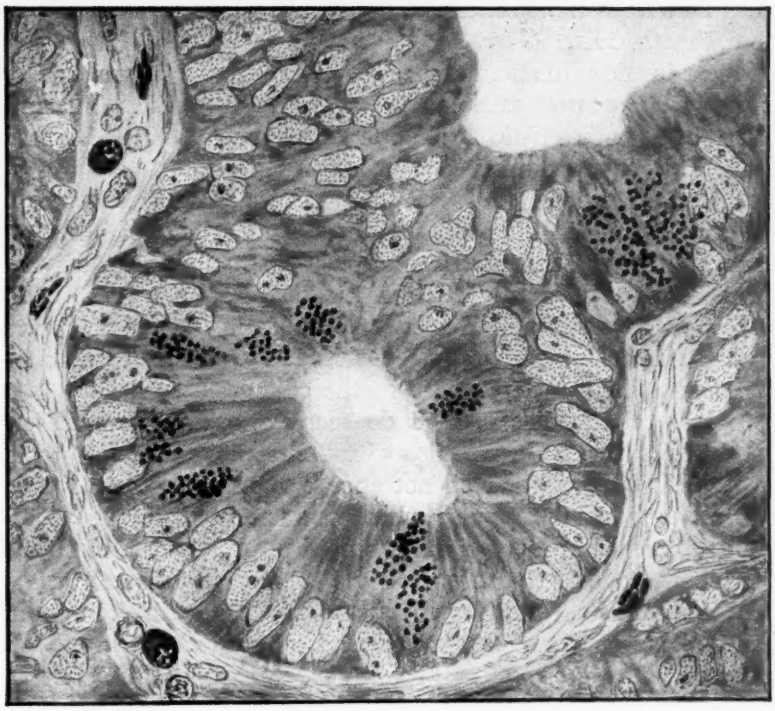


FIG. 442.—Projection drawing (A.C.L.) from papilloma showing selective staining of Paneth granules (granules and erythrocytes, bright green; plasma cells, deep red). Malachite green and acridine red. (× c. 800.)

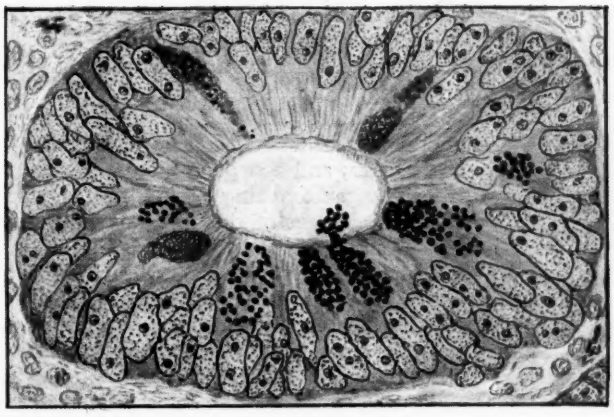


FIG. 443.—Projection drawing (A.C.L.) from papilloma showing varieties of distribution of Paneth granules, relation to goblet cells, discharge of granules, and intestinal type of free edge. Phosphotungstic acid hæmatoxylin and mucicarmine. (× c. 800.)

granule-containing cells are seen to be in mitosis. It is concluded from the shape of these cells and from the size, distribution, and colour reaction of the granules that they are Paneth cells. (*Figs.* 440–443.)

Also present in the epithelium are true goblet cells of the usual intestinal type; they are scarcely one quarter as numerous as the Paneth cells. The mucin gives a rather faint but characteristic coloration with mucicarmine, hæmatoxylin (of Delafield type), and celestin blue.

Neither Paneth nor goblet cells are present in the general wall except in those portions which lie between the roots of the papilloma. A very few cells of the tumour epithelium when stained with phosphotungstic acid hæmatoxylin followed by mucicarmine, or with Bensley's trichromic mixture, show Paneth granules in the deeper part with, towards the free edge, a transition to an almost homogeneous material giving the colour reaction for mucin. A still smaller number of cells shows the mucin-staining material deep to the granules.

After the use of Masson's method for sections, a very few cells are seen to have reduced the silver. These cells, which are all situated on the basement membrane, are small in size and do not appear to have any connection with the lumen; the silver-reducing granules which characterize them lie almost entirely in the infranuclear region. There seems no doubt that these are enterochromaffine cells (Kultschitzky, Ciaccio-Masson, 'yellow', 'basogranular' cells). No macrogranular leucocytes (Schollenleukocyten) are definitely identified.

This is a simple papilloma in the gall-bladder, covered by an epithelium which has the essential characters of intestinal epithelium, containing large numbers of Paneth cells, some goblet cells, and a few enterochromaffine cells.

Notes on Technique

With the exception of a loose fragment fixed in Zenker's solution (with 2 per cent acetic acid), the specimen was fixed in a Kaiserling's solution (pot. acetate 28 grm., chloral hydrate 27 grm., pot. nitrate 15 grm., formalin 150 c.c., water to 2000 c.c.).

Portions taken for histological examination were post-chromed by Kernohan's method[1] and after treatment with aqueous phenol solution[2] were brought through an ethyl-butyl alcohol series to butyl alcohol[3] and thence to paraffin. Of the stains mentioned, Reinke's neutral gentian was used as given under Bensley's neutral gentian in Carleton,[4] except that crystal violet was substituted for gentian violet (Conn[5]). Bensley's trichromic stain was used as given by Klein.[6] The phosphotungstic acid hæmatoxylin of Mallory was found particularly satisfactory as a stain for the granules, and a striking picture was seen after three hours' staining, the pre-treatment with permanganate being omitted. This form of staining, if followed by washing to reduce the red element of the hæmatoxylin, allows counterstaining although slowly with mucicarmine. Malachite green with acridine red is an easily used stain to show up the granules; this mixture, which Hitchcock and Ehrich[7] have proposed for the staining of plasma cells and Aschoff-body cells in tissues fixed by Zenker's fluid, has been found also to be very satisfactory for many other purposes, whatever the fixative, if the section is left for ten minutes in 1 per cent acetic acid, rinsed in water, and then stained as directed by the authors. With this stain the Paneth cells show bright green granules standing out against the soft pink of the cytoplasm; this affinity for the green is very marked, the granules retaining the stain even if the green be differentiated almost completely out of the nuclei.

The granules have been found to be Gram-positive, and an excellent picture is produced by Gram's stain following carmalum. Kirkpatrick's modification (unpublished) of the Gram-Weigert method for sections,[8] in which a sodium chloride solution replaces the Lugol's mixture, gives a very clear delineation of the granules, and appears, like phosphotungstic acid hæmatoxylin, to stain a higher percentage of the granules than the other stains used.

Some difficulty is experienced in comparing the results seen in different animals, since not only do different fixatives affect the staining of the granules and indeed also of the mucin, a point that will be further discussed below, but also the mucin of different animals may show different staining reactions and so make the recognition of the granules extremely difficult. This point was well brought out when carbol-thionin was used on Kaiserling-fixed mouse duodenum; here, with acetone dehydration, the mucin was pink and the Paneth granules dark blue; the same method when used on our tumour tissue showed the mucin retaining the blue stain and so failed entirely as a specific stain for the granules. Similarly with Kirkpatrick's stain the violet was almost completely removed from the mucin in the tumour tissue by aniline oil-xylol, but in formalin-fixed cat duodenum the mucin strongly resisted decolorization. Staining with celestin blue[2] for twenty minutes, followed by five minutes in fresh 1 per cent acetic acid, and then rapid dehydration, shows the nuclei blue, the mucin pink, and the granules unstained. Hamperl's[9] silver method did not seem to offer any advantage over the earlier method of Masson.[10] The most satisfactory staining of the granules seen with stains of the acid type was that produced by bichromate azo-eosin. This solution is prepared by mixing equal parts of 0·5 per cent aqueous solutions of azo-eosin and of potassium bichromate and filtering. Two minutes' staining is followed by differentiation in water and spirit.

THE SECRETORY ACTIVITY OF THE TUMOUR AND ITS RESULTS

RELATION TO MUCOCELE

Though the condition closely resembled that of mucocele in that the gall-bladder of a patient showing no jaundice was distended with a colourless fluid, it differed from mucocele in several respects. The fluid obtained when the gall-bladder was first opened was noted to be clear and watery, unlike the opalescent viscous content of a mucocele. Unfortunately no chemical analysis was made at this time. Further, the size of the gall-bladder exceeds that usually seen in mucocele. Judd and Castleton,[11] in 18,000 gall-bladders surgically removed, found only 35 with a long axis greater than 15 cm., and the largest which they record measured 20 cm. The long axis of the present gall-bladder after its removal measured 16·5 cm., and at the first operation when it contained 30 oz. of fluid, the gall-bladder must have been considerably larger; indeed, if proportional increase in its diameters is presumed, its long axis may be estimated to have measured 27 cm.

Nor did this gall-bladder behave during drainage in the manner of a mucocele. When a mucocele of the gall-bladder is drained without relief of the obstruction of the cystic duct, it is well known that the fluid drained diminishes rapidly in amount to a few cubic centimetres in twenty-four hours, and is of a viscid mucoid character. In this case the fluid output increased during the first eleven days to reach a peak at 154 oz., and during the last fourteen days was practically constant in amount at 65 oz. in twenty-four hours. Throughout the period of drainage the fluid maintained its watery character and showed a chloride content of about 0·8 per cent, a concentration which was considerably greater than that of the blood plasma (0·52 per cent NaCl).

Thus the condition was not one of mere mucocele but of distension by a watery fluid of high salt content presumably containing a small quantity of mucin. During the pre-operative stage this was evidently being produced at considerable pressure, while the amounts poured out after the institution of drainage show how vigorous was the underlying activity of the secreting surface.

SOURCE OF THE FLUID

The three possible sources of the fluid in the present case are the surviving gall-bladder epithelium, the areas of granulation tissue, and the epithelium of the tumour. In considering the first of these, we find that the only proved secretion of gall-bladder epithelium is that of mucin, most obvious in the condition of mucocele. In relation to sodium chloride and water the normal function of the gall-bladder is indeed absorption. Rous and McMaster[12] demonstrated that the gall-bladder concentrates the non-diffusible constituents of the bile at the expense of the diffusible without alteration of the osmotic pressure, which remains the same as that of the blood. This is effected by the almost complete removal from the bile in the gall-bladder of certain of the inorganic salts secreted by the liver, allowing the retention in the bile in greater concentration of bile salts and pigments, soaps, cholesterol, lecithin, and fats. Chabrol[13] similarly found that while the chlorine content of hepatic bile was 0·17 per cent, that of bile from the gall-bladder was only 0·005 per cent, a very marked reduction.

Even in a "white bile system" the chloride content of gall-bladder bile is relatively low. When obstruction of the common bile-duct occurs the retained bile is replaced by the colourless secretion of the bile-ducts and gall-bladder, constituting a "white bile system". The establishment of such a system is delayed in the presence of a healthy gall-bladder, presumably on account of its continued concentrating activity (Rous and McMaster[14]). Chemical investigation by Sjöqvist[15] of the contents of a white bile system associated with carcinoma of the pancreas showed a lower content of inorganic salts in the gall-bladder than in the common bile-duct.

Ravdin and others,[16] while studying the absorption of chlorides from the bile-free gall-bladder of the dog, showed that under certain artificial conditions the gall-bladder may actually secrete water and chlorides. Following the experimental introduction of saline solution at different concentrations, the osmotic pressure of the solution approached that of the serum within a few hours. Where hypertonic solutions were used, chlorides were absorbed, while at the same time water was secreted until isotonicity was reached. In a number of such experiments the establishment of isotonicity was succeeded by the secretion by the gall-bladder of a fluid containing 0·55 to 0·58 per cent of sodium chloride at a rate of less than 1 c.c. per hour. This the authors presume to have been due to damage to the gall-bladder wall by the hypertonic solution. In these cases chlorides occurred in the drainage fluid in the same concentration as in the blood, and the amount of fluid secreted never approached, even relatively, the quantities shown in *Fig.* 436.

We know of no circumstances, clinical or experimental, in which the gall-bladder has been known to secrete fluid comparable in constitution or amount to that in the present case.

Granulation tissue frequently replaces portions of the gall-bladder epithelium in chronic inflammatory conditions. Cholecystostomy in these cases is never followed by prolonged drainage of large quantities of fluid. Nor does granulation tissue in other situations produce fluid of this character or in this quantity.

It is therefore held that the main source of the fluid in the present case was the epithelium of the tumour. The significance of this will be considered along with the cytology of the tumour.

NATURE AND IMPLICATIONS OF THE FLUID LOSS

The clinical picture of dehydration which appeared a few days after the institution of drainage of the gall-bladder led to a more critical interest in the amount and constituents of the fluid lost externally. The outstanding features, to which reference has already been made, were its large quantity and high chloride content. Two estimations of the sodium chloride content of the fluid gave values of 0·79 and 0·81 per cent, this latter figure being obtained during a period when in fact dehydration was clinically manifest, urinary chloride practically absent, and the blood-plasma chloride low (0·52 per cent). It may be taken, then, that the chloride content of the fluid was fairly constant at about 0·8 per cent, and therefore the output may be calculated to have reached 35 grm. in the 154 oz. of fluid drained on the eleventh day, and during the later stabilized period to have been about 15 grm. in the 65 oz. of fluid per twenty-four hours. The tumour epithelium appears to have excreted essentially sodium chloride and water.

Massive loss of fluid and chlorides from the upper alimentary tract is most commonly seen in acute intestinal obstruction, but may also occur following the marsupialization of a pancreatic cyst (Janes[17]). The relative amounts of the various electrolytes lost in this way vary with the source of the fluid, so that different modifications of the acid-base equilibrium may be produced. Thus alkalosis is a recognized accompaniment of acute high intestinal obstruction, while in the external drainage of pancreatic juice acidosis may occur (Dragstedt[18]). In the present case an estimation of the alkali-reserve during a period of dehydration gave a normal value, which suggests that there was no marked alteration in the acid-base equilibrium.

The vast outpouring of sodium chloride and water by this tumour is in keeping with the observations (McIver[19]) on isolated loops of jejunum, that the upper part of the small intestine pours out a fluid of high chloride content. There was in this case dehydration and salt-depletion comparable to that of intestinal obstruction, without, however, any causative abnormality of stomach or intestine. The rapidity with which this clinical condition arose from the loss of what we may take to be "jejunal secretion", indicates the prominent part which may be played by the loss of intestinal secretion in the production of dehydration in intestinal obstruction. It also renders more explicable the occasional appearance of dehydration in the human subject prior to the onset of vomiting, and its constant occurrence in experimental obstruction in the rabbit, an animal where, through inability to vomit, large quantities of fluid accumulate in the stomach (Gamble and McIver[20]). In these cases we presume that the jejunum, like the stomach, continues to secrete a chloride-containing fluid which on account of the obstruction is functionally outwith the body, and no more available to that part of the bowel normally more absorptive than was the fluid drained to the exterior in the present case.

The prodigality of the tumour epithelium with chlorides is in marked contrast to the economy shown by the kidney. The tumour poured out chlorides continuously; the urine contained appreciable quantities only for a short time after each intravenous administration of saline. The state of the urinary chlorides, indeed, gave an earlier indication of chloride depletion than did the clinical condition of the patient. For this purpose the usual test-tube estimation with nitric acid and silver nitrate sufficed. Even during the periods when the clinical condition

resembled a severe intestinal obstruction, there was not the marked fall in urinary volume described in cases of intestinal obstruction (McIver[19]). Despite the gross fluctuations in water balance, the continued efficiency of the kidneys is apparent from the normal urea content of the urine and the blood.

The most striking fact in the case is that this tumour, not 3 *in. in diameter, concentrated sodium chloride from the blood plasma and poured it out at such a rate as to produce gross dehydration and chloride deficiency.*

DISCUSSION ON THE INTESTINAL CELL TYPES IN THIS TUMOUR

IDENTIFICATION OF THE PANETH CELL

The unusual histological picture already described has led to an extensive cytological investigation in this case. The literature on the Paneth cell is far from conclusive and almost entirely confined to studies on animals, so that the identification of the granular cells in this case necessitated some repetition of animal work to establish the validity of our technique. We hope to show that despite the unique site, true Paneth cells and enterochromaffine cells are present in this tumour and that, in so far as they are cell-types independent of one another and of the goblet cell, their presence is proof of the intestinal character of the tumour. The association in this case of Paneth cells with a profuse secretion will be considered as a possible clue to their little-known function.

The normal occurrence of Paneth cells has now been widely reported in the small intestine of vertebrates, but no record has been found of their presence in the gall-bladder. They have been reported in man as occurring throughout the small intestine and also (Oppel[21] and Clara[22]) in Brunner's glands. Patzelt[23] reports the finding in the human embryo of Paneth cells in those parts of the pancreatic and bile-ducts just adjacent to the ampulla of Vater. They are present in small number in the human appendix and cæcum, but probably not beyond that.

To prove the identity of our granular cells with those generally called Paneth cells, there is unfortunately no specific method. They, however, conform fully to the description given by Klein[6] and by Kull[24] and resemble the photographs in the latter's paper. Tehver,[25] faced with the same difficulty in proving that certain granular cells present in the Brunner's gland of the horse were Paneth cells, quotes Clara's statement[26] that Paneth cells have no definite characteristic other than their granularity. A further source of difficulty lies in the generally accepted fact that neither method of fixation nor type of staining reaction is capable of differentiating the granules of the Paneth cell from those of other granular cells such as the macrogranular leucocyte (Schollenleukocyte, Weill[27]) or the acidophil stage of the enterochromaffine cell. The eosinophil polymorph may occur in considerable numbers in the connective tissue and even occasionally between the epithelial cells, while the form of degenerate plasma cell which has circular acidophil vacuoles (Russell bodies) in its cytoplasm may also be present to provide a further complication. Of these different granular cells, the macrogranular leucocyte is the only one which is not an established type in the human intestine.

All the stains mentioned were tested on mouse jejunum, a site stated in the literature to be rich in Paneth cells. These cells were readily demonstrated, and

their reactions with every stain proved similar to those in the cells in question, although there was more irregularity in staining than in the tumour. The same stains were tested on cat jejunum. No granular cells resembling Paneth cells were seen in the crypts, but at the level of the neck of the crypts numerous granular cells were found in the epithelium; these cells are oval in shape and lie apposed to the basement membrane, not stretching out towards the lumen in the manner of Paneth cells; they differ also from Paneth cells in the circular shape and dark staining of the nucleus, and in the larger size of their granules. The granules, however, except that they are not stained by carbol-thionin, show an exact similarity in staining reaction with those of the Paneth cells. There seems no doubt that these cells are macrogranular leucocytes (Schollenleukocyten).

At the foot of the crypts in the jejunum of the cat and also of a pigeon (*Columba livia*) an occasional goblet cell was seen in which the mucin was definitely granular and the outer or almost expressed granules showed a marked affinity for the green in the acridine red - malachite green mixture, while the deeper mucin stained brown as in the mucin cells in other parts. The other staining methods, of which phosphotungstic acid hæmatoxylin seems the most reliable, confirmed the finding of Kull and others that there are no Paneth cells in the jejunum of the cat, and the finding of Baecker[28] that they are absent also from *Columba livia*. Observations on human operation material, which will be discussed under the heading of the occurrence of Paneth cells, further confirmed our use of the stains mentioned in the literature and also the validity and excellence of the three additional methods proposed as stains for Paneth granules.

The basis on which we can presume our cells to be Paneth cells lies in their shape and situation, and in the morphology, staining reactions and disposition of the granules.

Recognizable stages in the cycle of the Paneth cell have been suggested by Mols,[29] but, as he himself pointed out, the picture varies with the fixative used, a fact which has been emphasized by almost all workers on this subject. On comparing the effect of a series of fixatives (Hollande, formol-corrosive, Regaud, formalin, Kaiserling) on mouse jejunum, it is found that Kaiserling is an excellent preservative of the granules. Actually in the tumour tissue the percentage of granules staining poorly with phosphotungstic acid hæmatoxylin is considerably lower than in the correspondingly fixed mouse tissue. (The mouse was killed at mid-day without having received food that day.) Such granules in the tumour tissue as stain weakly appear to occupy no special position or relation in the cell. A further source of difficulty in justifying any conclusions based on the staining properties of the granules lies in the fact that some of the stains definitely colour a higher percentage of the granules. In view of these difficulties and the uncertainty in the literature, there seems no possibility of definitely correlating the histological picture with any stage of secretory activity on the part of the Paneth cells.

The normal position of the Paneth cell is strongly in favour of its being secretory. In man it occurs mainly in the small intestine, where a large and important secretion is poured into the lumen; its presence in very small numbers in the human cæcum is not a counter-argument, since a small amount of secretion is produced in the large intestine.[30] Its situation in the crypts and not on the villi is likewise in favour of an active or secretory function, as is the often observed discharge of the granules. A further argument is found in the present case: the

large amount of fluid has already been shown to be the product of the tumour epithelium, and so either the Paneth cells or the non-granular cells, whether or not they are different stages of the same cell, must be the source of this fluid.

THE PANETH CELL AND THE GOBLET CELL

The relationship of the Paneth cell to the other types of normal intestinal cell has been deduced mainly from observations on the lower animals. Clara,[26] from work on avian material, considers that it is normally a type *sui generis*, and many workers concur in this view. Bizzozero[31] considered the Paneth cell a young mucin cell, while Kull,[24] supported by Chuma,[32] considers that it is a regressing mucin cell. Our findings certainly suggest a possible relationship between the goblet and the Paneth cell, in so far as we have found in different portions of the same cell the colour reaction for mucin, and also typically stained Paneth granules. This transitional type is well shown in the sections stained by phosphotungstic acid hæmatoxylin and mucicarmine. Bizzozero considered that in the cycle, the Paneth granules become smaller and then a mucin reaction develops at the free end of the cell. In most of our transitional cells the appearance conforms to his illustrations but in a few the relative disposition of mucin and Paneth granules is reversed.

Clara,[26] in stating that the Paneth cell is a type *sui generis*, is apparently fully cognisant of the mucin-like staining in some Paneth cells; he points out that mucin-stains are not entirely specific and states that variations in this can depend on the fixative used. This fact was well seen when staining mouse intestine by Kirkpatrick's stain; tissue fixed in Kaiserling allowed easy decolorization of the mucin, and so was satisfactory for observing the Paneth cells, but after fixation in formol-corrosive, Hollande's or Regaud's fluid, the mucin held the violet almost as strongly as does cat mucin. Baecker[28] more recently has again pointed out the basic staining of Paneth granules after picric acid fixation, a finding which is explained in some measure by Boerner-Patzelt,[33] through a study of the iso-electric point of the Paneth granules after different fixatives. Thus, in a critical examination of the transitional type of cell, both fixative and stain demand scrutiny. From work on mouse tissue it appears that formalin solution (sodium chloride 10 grm., sodium sulphate 30 grm., formalin 200 c.c., water 800 c.c.) or Kaiserling's solution is not only a better fixer of the granules than is formol-corrosive, Hollande's or Regaud's fluid, but is also a better mordant, in that the granules stain more uniformly. On the tumour tissue Kaiserling's solution likewise showed both these advantages over Zenker's fluid. It may therefore be justifiably presumed that the gall-bladder was fixed unwittingly in the most suitable fixative for Paneth granules. The phosphotungstic acid hæmatoxylin solution (naturally ripened) after three hours' staining showed the granules of a deep purple colour, and with subsequent treatment with water and spirit there was little or no evidence of any staining of the mucin. For optimum although slower staining the sections should be pre-treated with a reducing agent such as potassium metabisulphite. Southgate's mucicarmine was used as the stain for mucin, and from staining of animal intestine it would appear that the phosphotungstic acid hæmatoxylin beforehand does not deviate the specificity of this stain. It is therefore held that if cells exist which contain both mucin and Paneth granules, then the technique outlined above is optimal for their exhibition. The fact remains, however, that mucicarmine also stains cartilage and tissue

which has undergone myxomatous degeneration, and so a substance may be present in Paneth cells which stains red and is yet not mucin.

Also against a relationship between the two cell types is the observation that various stages of development up to discharge of the cell contents are seen in both the Paneth and the mucin cell; further, the rarity of the transitional type is remarkable if the numerous Paneth cells in this tissue are really related to the far from infrequent goblet cells. The relationship between these two cell types remains an open question.

Goblet cells do not occur, according to Maximow,[30] in the normal human gall-bladder. They have, however, been reported in the extrahepatic biliary ducts of man, mainly in "the intestinal portion of the ductus choledochus" (Macklin[34]), and are present in the gall-bladder of some animals (Macklin).

Whatever the relationship between goblet cell and Paneth cell in the normal intestine or even in this tumour, the actual presence of goblets may be considered at least confirmatory of the possibility which the Paneth cells suggest, that the epithelium of the tumour is truly intestinal in type.

THE PANETH CELL AND THE ENTEROCHROMAFFINE CELL

The silver-reducing cells in the tumour have the situation, the appearance, and that type and disposition of their granules which justify our recognizing them as enterochromaffine cells. The presence of these cells is of interest in view of the existing controversy as to their origin.

Cells of this type have been reported by Tehver[35] in the gall-bladder of the cow, but no record has been found of their existence in the human gall-bladder (quoted Hanser[36]), although Patzelt[23] has found them in those parts of the pancreatic and biliary ducts just adjacent to the ampulla of Vater in the human embryo, where he also described Paneth cells. All our enterochromaffine cells show a very definite localization of the granules in the basal part of the cell, a disposition which, according to Hamperl,[9] is very marked in the normal human duodenum.

The number of silver positive cells seen in the tumour sections is small, but, as Masson[10] points out, the silver reaction is capricious when applied to sections. According to Macklin, the enterochromaffine cell granules degenerate more rapidly than those of the Paneth cell and so the ratio of the two types as seen is possibly not a true index. The tissue, however, was in fixative solution within half an hour of removal, and Hamperl[9] estimates that it is four or five hours before the granules lose their silver-reducing power.

Enterochromaffine cells are few, according to Tang,[37] in those animals in which Paneth cells are numerous, as in the squirrel; the opposite condition also holds good, as is typically seen in the pig. Whether this numerical relationship, which is accepted by other authors, signifies any functional relationship is a very open question. Kull[38] is quite definite that no possible relationship can exist, basing his argument on the findings in those animals in which Paneth cells are very few or absent. Cordier's work,[39] in which under the influence of pilocarpine both enterochromaffine and Paneth cells are seen to lose their granules, suggests that they are both entodermal secretory cells, but Macklin[34] some years later still considered that there was no real evidence that the enterochromaffine cells are in any way concerned with digestion or absorption. The evidence from the literature

does not seem to show much in favour of a relationship between the Paneth cell and the enterochromaffine cell, nor does it give any satisfactory proof of an entodermal origin for the enterochromaffine cell.

Normally the enterochromaffine cell is confined in the human subject to the alimentary tract proper and has never been recorded in the gall-bladder; whichever view is held as to the origin of this cell, its occurrence in the tumour epithelium but not in the general gall-bladder wall is significant evidence of the intestinal nature of the tumour.

We have therefore in this gall-bladder tumour three intestinal cell-types capable of histological identification, two of which have never been reported in the human gall-bladder.

THE PANETH CELL IN PATHOLOGICAL CONDITIONS

The relationship of this cell to pathological processes is apparently little known, but the scanty evidence from which it has been assumed by some that Paneth cells are increased near a focus of irritation must now be examined. The occurrence of Paneth cells in the intestinal islets of the stomach will be discussed later.

Schmidt,[40] who states that Paneth cells are practically absent from the large intestine, found them in three cases. Two of these were of simple polyp with Paneth cells in the epithelium of the growth itself. The third case was of carcinoma, and Paneth cells were noted in the otherwise apparently normal epithelium immediately adjacent to the malignant transition. Unfortunately there is no note of the part of the large intestine involved. Eklöf[41] reports the presence of numerous Paneth cells in the neighbourhood of a carcinoma in the transverse colon, and this would appear to be the one substantiated case in which Paneth cells have been recorded in the actual epithelium of the large intestine distal to the cæcum in the human adult. His only other observation was made on large intestine from an operation for strangulated hernia, but in this he found no Paneth cells. The implication drawn from this work has been that Paneth cells are increased in the neighbourhood of a focus of irritation. With a view to testing the truth of this, some pathological and normal operation material was examined after staining by phosphotungstic acid hæmatoxylin. The sites and results are seen in *Table I*.

From *Table I* it will be seen that Paneth cells were found in large numbers, actually two to seven per crypt and in five out of six crypts, in the small intestine and in a perfectly healthy Meckel's diverticulum. An occasional Paneth cell was seen in the cæcum and in the appendices examined, but none in the remainder of the adult large intestine. The available portions of normal epithelium unfortunately do not make a complete control, but from one of the resections for carcinoma of the cæcum (*Case* 4), ileal mucosa was available up to 15 cm. from the still patent valve, and cæcal mucosa to 5 cm. distal to the tumour. In a comparison of the cæcal portions there was a very slight increase in the number of Paneth cells toward the growth; since the general view is that Paneth cells occur in the cæcum and not in the remainder of the colon, this gradation may be taken as normal. In examination of the 15 cm. of ileal mucosa, there appeared to be no variation in the number of Paneth cells except in the $\frac{1}{2}$ cm. immediately adjacent to the malignant change where Paneth cells actually were fewer. This diminution in the boundary zone was also seen in *Case* 5. In no case were Paneth cells or any cells resembling them

seen among those frankly carcinomatous, although Schmidt[40] quotes Saltykow[42] as finding Paneth cells in a few alveoli of an adenocarcinoma of the stomach.

Of the rectal papilomata from the child, one showed a single but quite definite Paneth cell (*Case* 18); the other (*Case* 19) showed none, but in this case the surrounding mucosa showed an interesting appearance. The epithelium, differing markedly from the tumour cells which are almost entirely chief cells, consists of the usual goblet cells, but in the deeper parts of the mucosa the crypts are continuous with glands of the 'pyloric' type. These are morphologically similar to

Table I.—Site of Paneth Cells in Nineteen Cases

Case	Site	Paneth Cells	Case	Site	Paneth Cells
1	Normal jejunum ..	+++	10	Splenic flexure near carcinoma	—
2	Epithelium near infected diverticulum	+++	11	Splenic flexure near carcinoma	—
3	Ileum near sarcomatous ulceration	+++	12	Sigmoid colon near carcinoma	—
4	Ileum 15 cm. from carcinoma at valve	+++	13	Sigmoid colon near carcinoma	—
5	Ileum 8 cm. from carcinoma at valve	+++	14	Normal colon, colostomy	—
4	Ileum adjacent to carcinoma	+	15	Normal colon, colostomy	—
5	Ileum adjacent to carcinoma	+	16	Rectum near carcinoma	—
6	Meckel's diverticulum	+++	17	Rectum near carcinoma	—
7	Inflamed appendix	+	18	Papilloma of rectum (child)	+
8	Inflamed appendix	+	18	Adjacent epithelium	—
9	Inflamed appendix	+	19	Papilloma of rectum (child)	—
4	Cæcum near carcinoma	+	19	Adjacent epithelium	++
5	Cæcum near carcinoma	+			

the type seen in the deeper parts of the Aschoff-Rokitansky sinuses (Nicholson[43]) and are likewise negative to mucicarmine (formalin fixation). In the depths of the crypts, whether ending normally or in continuity with the 'pyloric' glands, there are numerous Paneth cells in the epithelium. An occasional Paneth cell is seen among the clear cells at the region of transition from goblet cells to clear cells.

Comparison with animal tissues is of little value as a guide to the normal condition in the human since the distribution varies so greatly. Trautmann[44] finds the condition in the horse and ass to be similar to that accepted for man,

whereas Hamperl[45] finds that in the mole and the bat Paneth cells are present in the large intestine almost to the anus.

It seems reasonable to conclude that in the human adult Paneth cells are numerous in the small intestine, scanty in the appendix and cæcum, and absent from the remainder of the large intestine. *There seems no reason for believing that a focus of irritation in any part of the adult intestine will lead to an increase of Paneth cells.*

HETEROTOPIA OR METAPLASIA ?

Having stated our belief that this tumour is formed of intestinal epithelium, we are now faced with the problem of how this arose in the gall-bladder. The process of metaplasia has been so often invoked to explain the intestinal islets in the stomach that its possible application to our case demands examination. We hope to show, however, that the specialized character of the cells present makes heterotopic misplacement the only acceptable solution.

Metaplasia " occurs when a differentiated tissue loses its characters and assumes those of another differentiated tissue."[46] The transition is generally towards a more protective form, and though this may be to a moderately specialized type as seen in the evolution of goblet cells from the epithelium of the extroverted urinary bladder, the usual association with mechanical or inflammatory irritation makes it understandable that there occurs no high degree of morphological or functional evolution.

In the heterotopic islands believed to be due to a dislocation of tissue during the period of development, a visibly complete perfection may be seen, and the peptic ulceration produced near gastric islets in a Meckel's diverticulum is one of the proofs that these areas may have a function corresponding to their appearance.

Metaplasia as usually seen in the gall-bladder shows an epithelial change in association with stone to the squamous form, or the appearance in the chronically inflamed viscus of Aschoff-Rokitansky sinuses with, in their deeper parts, areas of cells resembling ' pyloric ' glands.[43] Heterotopia in the gall-bladder has been reported by von Hedry,[47] who describes a case of accessory pancreas in the wall of the gall-bladder, while adenomyomata have been recorded in the wall, which, in the complete absence of signs of inflammation, have been considered as ' hamartomata '.[48] Egyedi[49] has recently described a papilloma of gall-bladder in which the epithelium was of gastric type, but no record has been found in the literature of heterotopic intestinal epithelium in the gall-bladder.

The commonest form of abnormally situated intestinal epithelium is that seen as islets in the stomach. Chuma[32] found areas of intestinal epithelium in the stomach of 15 of a series of 73 unselected autopsy cases. Paneth cells were present in this intestinal epithelium in 4 of these cases. In his observations on operation material, he noted intestinal islands in the stomach of 28 (70 per cent) out of 40 cases ; in all of these there were Paneth cells. These islands were in each case associated with or near a lesion, ulcer, cancer, or chronic gastritis. According to Chuma, who reviews the German literature on this subject, intestinal epithelium is never found in a normal stomach. Borrmann[48] later supports this view, and goes so far as to say that the intestinal islets occurring in the stomach of older adults must be considered as always due to a pathological condition of the viscus. Moszkowicz[50] explains the occurrence of the islets on the basis of differentiation

of an indifferent epithelium growing up in an irritated area, and uses for this the term ' indirect metaplasia '. Clar,[51] on the other hand, brings forward evidence from study of fœtal stomachs and of those parts of gastric resections well away from the lesion, in no case chronic gastritis, all of which goes to suggest that these islets are congenital displacements. Hurst and Stewart[52] also report the presence of intestinal islets in otherwise normal stomachs. The nature of the process leading to the presence of intestinal islets containing Paneth cells in the stomach is therefore not yet conclusively defined.

If the view were correct that these islets are due to metaplastic change, then the type of epithelium in the islet, however closely it resembled that of the small intestine, would require to be considered a de-differentiated form of gastric epithelium. There is no doubt that Paneth cells occur very commonly in these islets and would therefore have to be placed in the category of the less specialized alimentary types ; they could then be considered a possible appearance in the debasement of any specialized alimentary epithelium, including that of the gall-bladder. We have attempted, on the other hand, to show that the Paneth cell is a specific cell-type structurally and functionally, which is not increased in the alimentary tract or produced in the gall-bladder in response to irritation. We therefore consider the presence of Paneth cells to be strong evidence in favour of the heterotopic nature of the present tumour.

The goblet cell lends no definite help to the argument. This type is present in our tumour and in the intestinal islets of the stomach, whereas normally it is not found in either gall-bladder or stomach. Thus it would seem to bear the same relationship to the question as does the Paneth cell. The frequency, however, with which goblet cells appear in the chronically inflamed gall-bladder without the appearance of other intestinal forms, suggests very definitely that they arise here by a process of metaplasia. This being so, we are unable to utilize their presence in our tumour as evidence that the epithelium is of fully formed intestinal type, even though this may well be the true explanation of their presence.

The finding in our tumour of enterochromaffine cells is of particular interest since it is a type which, so far as we know, has never been found in the human gall-bladder. There seems no doubt that this is a specialized cell, and, though its presence here is surprising if one accepts the hypothesis that the enterochromaffine cell has its origin outwith the entoderm, the finding of this cell definitely favours the view that this epithelium is not the product of metaplastic change.

It is therefore concluded that the epithelium of this tumour is truly intestinal, and sufficiently specialized in morphology and function to be explicable only on the view that the tumour arose in an area of heterotopic epithelium.

PAPILLOMA OF THE GALL-BLADDER: REVIEW OF PUBLISHED CASES

The statement that papilloma of the gall-bladder is a rare lesion introduces the subject in most of the standard works of pathology and surgery. A study of the literature has suggested that, even in some of the cases most frequently quoted, the diagnosis has been too readily accepted.

Much confusion has arisen within the last twenty years through the application of the term ' papilloma ' to lesions which many are unable to accept as in any true

Table II.—Cases Reported as 'Papilloma' of the Gall-bladder

Author and Date	Features	Suggested Diagnosis
Heschl,[58] 1852	Localized polypoid fat-laden projections; stone present; character of probable secondaries in liver not confirmed microscopically	Carcinoma
Klob,[59] 1856	Two cases. Localized polypoid projections; inflammation; stone present	—
Rokitansky,[60] 1861 ..	Circumscribed polypoid lesion	—
Mettenheimer,[61] 1871 ..	Fat-laden papillæ; bile in gall-bladder; no stones	Cholesterosis
Schüppel,[62] 1880 ..	Small gall-bladder filled by papillary mass attached over a wide base ('myxopapilloma'); no bile in gall-bladder; no stones	Papilloma
Birch-Hirschfeld[63] 1895 (?)	Circumscribed polypoid lesion	—
Ringel,[64] 1899	'Multiple papillary fibromata' considered to be possibly early carcinoma, but patient was well 4 years later	Cholecystitis polyposa
Hansson,[65] 1905 ..	Enlarged gall-bladder filled by pus, stones, and a degenerate papillary mass the size of a fist	Papilloma
Pels-Leusden,[66] 1906 ..	Cholecystostomy; generalized papillary formation; high cylindrical epithelium. Two years later similar growth at mouth of fistula. Death three years thereafter from phthisis; stone in common bile-duct; no secondary tumours	Papilloma
Sand and Mayer,[67] 1910	Mucocele relieved by removal of enlarged cystic gland; no stone; generalized papillary formation (papillæ 2 mm. long); goblet cells present. "An exaggeration of the normal delicate villi"	Cholecystitis polyposa
Dominici,[68] 1911 ..	Mucocele; generalized papillary growth (papillæ 5 mm. long); goblet cells present	Cholecystitis polyposa
Schoenlank,[69] 1914 ..	Enlarged gall-bladder with papillary projections; epithelium many layered; bile-coloured mucin in gall-bladder; stone previously passed. Second operation—papillary carcinoma in left lobe of liver. Squamous epithelium alongside an area of ulceration in the gall-bladder	Carcinoma
Kaufmann,[70] 1922 ..	Three cases. Each had several small tumours; no detail of size; no histology; accepts Sand and Mayer's case and this lesion is therefore presumably comparable	Cholecystitis polyposa

Table II.—CASES REPORTED AS 'PAPILLOMA' OF THE GALL-BLADDER, *continued*

AUTHOR AND DATE	FEATURES	SUGGESTED DIAGNOSIS
Landsberg and Zaorski,[71] 1923	Cholecystectomy for chronic cholecystitis; gall-bladder contained bile and, in the fundus, a stalked papilloma; no histology in abstract	? Papilloma
Abell,[72] 1923	Two cases. Multiple papillary outgrowths One case. Operation for acute cholecystitis; gall-bladder filled with villous growth considered malignant (no histology); cholecystostomy. Three years later, operation for common bile-duct obstruction; no sign of gall-bladder; non-malignant papillary adenoma in common bile-duct, thought to be possibly a transplant from the original growth	Cholecystitis polyposa Papilloma
Risak,[73] 1927	Polypoid adenoma lying free in gall-bladder, size of hen's egg; no stone; too necrotic for histological study	? Polypoid adenoma
*U.C.H.,[74] 1929	Annular villous papillary growth 1·5 × 0·5 in.; single stone; delicate fibrous tissue processes with myxomatous change covered with a layer of columnar epithelium of gall-bladder type; goblet cells present; no Paneth cells	Papilloma
Vecchi,[75] 1929	Papillary tumour the size of a hen's egg; histologically complex villous papilloma with tall columnar epithelium, single layer with 'border' at free edge. No goblet cells in tumour; gall-bladder contained bile but no stones	Papilloma
Henry,[76] 1933	Sessile growth, 2·5 cm. wide and several smaller isolated growths; also one in cystic duct and one in common bile-duct. Histologically—simple papilloma	Papilloma
Egyedi,[49] 1934 ..	Operation for chronic cholecystitis; mucocele with single stone and tumour at neck. Histologically—papilloma consisting of gastric epithelium including chief and parietal cells	Papilloma of heterotopic epithelium
*R.C.S. Edin. (unpublished)	Obstructive jaundice with colic; cholecystectomy; numerous small stones in cystic duct; gall-bladder contained soft papillomatous material (*a*), and had a thick irregular wall (*b*). Histologically: (*a*) Somewhat degenerate villous papilloma; no apparent Paneth cells. (*b*) Carcinoma	Carcinoma

* We are indebted to Professor C. C. Choyce and the University College Hospital Medical School, and to the Royal College of Surgeons of Edinburgh, for details of these cases, for the opportunity to stain sections, and for permission to publish our findings in this paper.

sense neoplastic. Thus Mayo[53] states that in papillomatous cholecystitis the ' papillomas ' vary from twice to five or six times the length of normal villi and are laden with a fatty substance responsible for their yellowish appearance ; MacCarty[54] goes further in saying that these papillomas are histologically identical with the villi in the strawberry gall-bladder, being merely larger and branched. These writers are obviously describing the familiar cholesterol polyp, and, as Graham[55] has suggested, a clear differentiation should be made between this condition and true papilloma. Phillips,[56] who with Mayo applies the term ' papilloma ' to the cholesterol polyp, describes in addition a sessile papilloma due to infection ; his account of the gross and microscopic appearances of this and the associated conditions suggests only the proliferation commonly seen in chronic cholecystitis. Nothing is gained by attributing to this lesion a name which implies that it is essentially neoplastic, and it would seem preferable to use the term ' cholecystitis polyposa ' or, as suggested by King and MacCallum,[57] ' cholecystitis glandularis proliferans.'

The small pedunculated growths sometimes described as papillomata, but histologically devoid of villous formation and containing a high proportion of connective tissue, are probably to be looked upon as adenomata comparable to those occurring within the wall.

A further source of confusion in nomenclature occurs in the earlier cases in the German literature, where Heschl[58] and Klob[59] describe under the title of ' Zottenkrebs ' what they themselves considered to be simple lesions.

The cases shown in *Table II* have been published or quoted as papillomata of the gall-bladder.

The classification of these cases has been rendered difficult by the lack of available detail, particularly in the earlier publications. On these grounds we do not venture to place in any definite category the cases of Klob, Rokitansky, Birch-Hirschfeld, and Landsberg and Zaorski. Mettenheimer's case we consider to have been one of cholesterosis with cholesterol polyp.

In the cases of Ringel, Sand and Mayer, Dominici, Kaufmann, and Abell (first two cases), the lesion was multiple or generalized, the outgrowths were small, and evidence of cholecystitis was present, though sometimes only in the form of stones or goblet cells. Here we consider the diagnosis to have been cholecystitis polyposa. Risak's case was probably one of polypoid adenoma.

The cases of Heschl, Schoenlank, and R.C.S. Edin. were definitely malignant. The papilloma of heterotopic epithelium described by Egyedi has already been mentioned along with our own comparable tumour.

The remaining cases, those of Schüppel, Hansson, Pels-Leusden, Abell (third case), U.C.H., Vecchi, and Henry, were, as far as we can estimate from the descriptions, discrete villous growths of considerable size projecting from the mucosa of the gall-bladder. The presence of subsidiary growths on adjacent portions of the gall-bladder wall, in the cystic duct, in the common bile-duct, or in a post-operative fistula, seems quite in keeping with a diagnosis of papilloma. Histological description, where available, was of a simple outgrowth of gall-bladder epithelium with a thin much-branched stromal core. In agreeing with Hanser[36] that the differentiation between papilloma and chronic proliferative cholecystitis is usually difficult, and while admitting that the inadequacy of description of many recorded cases makes retrospective classification insecure, we yet feel

that the cases in this last group do justify their classification as true papillomata of the gall-bladder.

Of the seven cases noted, three show simple transplantation, which, along with their villous structure, recalls the papillomata occurring within the urinary system. In the two recent malignant cases already mentioned, a comparatively simple villous papilloma was associated with adenocarcinoma of the gall-bladder wall. In Schoenlank's case the epithelium of the papillomatous portion was in part two-layered, but the appearances were virtually those of a benign tumour except at the base, where gradual transition was seen to frank malignancy with infiltration. In the R.C.S. Edin. case the available parts of the papillomatous portion show a hyperplastic but not definitely malignant epithelium. At the base of the tumour there is obvious carcinoma. From the description in Schoenlank's paper and the appearances in the R.C.S. Edin. case it seems reasonable to compare these tumours, as Illingworth[77] has recently done, with those villous papillomata of the urinary system which show so-called ' malignant transformation '.

Thus the cases accepted as papillomata of the gall-bladder fall naturally into three groups: (1) Simple villous papilloma; (2) Simple villous papilloma with transplantation; (3) Villous papilloma with ' malignant transformation '. It is noteworthy that this classification of the papillary tumours of the gall-bladder is identical with that frequently employed in the description of the corresponding lesions in the urinary system. The inter-relationship of these three groups is no further elucidated by our study, but the underlying connection would seem on histological grounds to be the same in the two situations.

Apart from the case of Egyedi, the epithelium of all the tumours found has been described or presumed to be of gall-bladder type. In no case has a tumour of intestinal epithelium been described. The only variation noted has been the presence of occasional goblet cells in the epithelium of the tumour in the U.C.H. case, but, as in the chronically inflamed gall-bladder, these occur merely as isolated units in an otherwise typical gall-bladder epithelium.

SUMMARY

An unusually distended gall-bladder causing no symptoms other than those due to its size was surgically drained. Thereafter surprisingly large volumes of fluid poured out from the wound. The fluid proved to be sodium chloride and water, at a concentration considerably higher than that of the blood plasma. Dehydration and chloride depletion resulted within five days and were successfully treated by intravenous administration of saline. The continued outpouring of fluid, which during one period of twenty-four hours reached $7\frac{3}{4}$ pints, necessitated the repetition of this treatment at intervals of five or six days throughout the thirty-four days of drainage.

The gall-bladder was then removed and found to contain a large papilloma. Histological examination showed the epithelium of this tumour to resemble that of the small intestine, with Paneth cells, enterochromaffine cells, and goblet cells. The source of the fluid is considered to be this intestinal epithelium, and the question is raised of the possible part played by intestinal secretion in the dehydration and chloride depletion of acute high intestinal obstruction. Estimation of the

urinary chlorides is advocated as providing an early indication of chloride depletion in comparable cases.

Histological investigation of the cell types in the tumour led to the conclusion that the Paneth cell, the enterochromaffine cell, and the goblet cell are independent types and that the presence of the first two, which have never been previously reported in the gall-bladder, proves that the epithelium is truly intestinal in character.

The specialized nature of the cells in this papilloma is held to show that the tumour arose in an area of heterotopic intestinal epithelium. A brief critical analysis is given of cases previously published as papilloma of the gall-bladder, including one papilloma of heterotopic gastric epithelium.

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THE TREATMENT OF ACUTE INTRAMAMMARY ABSCESS, BY INCISION AND BY ASPIRATION

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An account is here given of acute intramammary abscess with particular reference to treatment, and the statements we make are founded partly on the literature and partly on our own observations. We have recently been in the position to observe every case of breast abscess treated at St. Thomas's Hospital over a period of about a year. It has been possible to collect 71 cases, and careful records have been kept of each case. Some of these patients were treated in the Out-patient Department, but those that failed to improve, and those in which suppuration supervened during the puerperium, were admitted to the Septic Block, where a certain number of beds were always available.

ETIOLOGY

It is not unusual to find suppuration in the non-lactating breast. In a series of 102 cases, Bryant[11] (quoted by Sampson Handley) found an incidence of 20·5 per cent in cases that were neither pregnant nor lactating. In our own series there were 11 cases, giving a percentage incidence of 15·5.

The abscess occurring in the non-lactating breast is of little clinical importance, apart from its occasional association with carcinoma. The responsible organism appears to be the *Sta. aureus*, the path of entry being either from the nipple or from the blood-stream. There appears to be no direct evidence in favour of either, as in our own series the nipple appeared to be perfectly normal and there was no evidence of septic foci elsewhere in the body. As in most pyogenic infections, a lowered general condition, resulting probably from social conditions, appeared to be the underlying factor. However, the prognosis in these non-lactating cases is good.

The abscess occurring during lactation presents a different picture. The organism concerned appears to be either a *Sta. aureus* or a streptococcus. The latter we believe to be very rare in breast abscess without secondary infection. In no case of our series in which the pus was cultured was a streptococcus grown. We therefore find ourselves in agreement with both Benians[1] and Moon and Gilbert,[19] in considering the streptococcus to be rarely a causative organism. The organisms are present in the milk from normal breasts,[12] and in children's mouths.[13] The actual portal of entry is debatable. Benians has gone so far as to distinguish between two types of abscess: (1) That in which entrance is effected by streptococci via cracks in the nipple; and (2) That in which staphylococci find access to the breast by means of the ducts. Hamilton Bailey[3] has confirmed this distinction.

The presence of cracked and sore nipples undoubtedly plays an important part in the etiology. Ehrlich[5] found 24 per cent of cracks on macroscopical examination

of 84 cases. Kehrer[4] considered that of all cases of cracked nipple, 25 per cent developed mastitis. In 60 consecutive lactating breasts in which infection had supervened, we found 17 cases with a definite history of cracks and a further 19 cases with painful or 'sore' nipples. This gives 36 cases with a history of nipple trouble, an incidence of 60 per cent in all.

Sudden weaning or death of the infant, with its attendant breast engorgement, appeared to be a factor in 10 per cent of our cases.

Pain is the outstanding clinical feature of a cracked nipple. Owing to pain the mother cannot tolerate prolonged feeding on the breast, and unless the condition is bilateral, will prefer to feed the infant on the other side. In consequence of this the affected breast remains relatively engorged.

We believe that the essential action of a cracked nipple lies in the engorgement it produces and not in its action as a path of entry for organisms. It follows from this that the treatment of any nipple crack becomes of the highest prophylactic importance, a point already emphasized in the literature.[2] (In a recent paper the importance of nipple trouble has been discountenanced.[19])

PROPHYLACTIC TREATMENT

1. Before Engorgement.—There is no need to mention the steps to be taken in maintaining the integrity of the nipple, for these are repeated and emphasized in every text-book on infant feeding; but one cannot help being struck very forcibly by the utter disregard of ordinary principles that should be employed once a nipple has actually become cracked or sore. These principles should be:—

a. Scrupulous cleanliness, without which all other treatment is surely useless.

b. The employment of hardening material, in the nature of Friar's balsam and perchloride of mercury.

c. The use of a shield for protection of the nipple at the first evidence of soreness, without awaiting the presence of a macroscopic lesion.

d. The use of a breast pump. This instrument is absolutely invaluable for use on the affected side, even where a shield is already in use, to make certain that the breast is completely emptied after feeding. The breast pump is condemned by some, since they consider that suction merely increases the output from the affected breast and thus produces the very engorgement to be avoided. This is a fallacious argument. Firstly, the breast pump is less stimulating than the normal direct feeding, and, secondly, it is always capable of draining any increased output for which it may have been responsible.

e. Where a shield fails to relieve pain, no further time should be wasted in direct feeding on the breast concerned. At the best simple engorgement will result. There should be no hesitation in removing the infant from the side in question and feeding it indirectly, the breast being completely evacuated with the breast pump, and the milk being administered by means of a bottle.

When the nipple has settled down these steps mentioned above are reversed until the baby is feeding directly on the unprotected nipple without the production of pain and engorgement.

2. After Engorgement.—Engorgement of the breast appears to be of two distinct types:—

a. A diffuse engorgement of sudden onset. Constitutional signs are marked.

There is usually a temperature of 102° to 103°; the pulse is rapid, the tongue furred, the patient feels ill and complains of a headache, lassitude, etc. On examination of the breast the organ is diffusely enlarged, usually very tense, tender throughout, and the overlying skin is flushed and hot. It is doubtful if any improvement in the condition is made without active treatment. With active treatment the condition will often settle completely in a few days, or else pass to the local type of engorgement now to be described.

b. In the local type of engorgement (which may occur spontaneously during lactation, or follow directly during treatment of the diffuse type) constitutional symptoms are similar but less marked. There is a localized tender mass in the breast, the skin over this is red, and the condition is of practical interest in that it may easily be confused with that of localized suppuration.

The treatment of the diffuse type of engorgement is simple and effective. The patient is put to bed when practicable. Local heat may be applied with thermogene wool, antiphlogistine, a linseed poultice, or a hot bottle, and the nipple should not be included in the dressings as it tends to lose both its shape and its firmness. The effect of hot fomentations is too transient to be of value. Hot bathing before attempting to evacuate the breast is helpful, and the breast must be supported between feeds. If the nipple is normal (unusual in these cases), the baby should be fed from the other breast only and the affected breast carefully emptied after feeding. Should there be any pain from direct nipple feeding, then the breast pump must be employed instead. (Attempts at expression are strongly contra-indicated on account of the intense pain produced from pressure on an already tender breast.) Evacuation should be attempted at frequent intervals, at least every three hours, and if possible every two hours. Should this treatment fail, it will usually be because even the breast pump produces intolerable pain on pressure, and cannot be employed satisfactorily.

With regard to the localized type of engorgement, the treatment is from first to last the employment of the breast pump. Local application of heat is unnecessary and appears to aggravate the condition. Rest again is essential, and a breast support is valuable in relieving pain. (It cannot be too strongly emphasized that such support must lift up the breast without compressing it against the chest wall.)

Expression of milk is dangerous in this condition as it is impossible in many cases to tell whether suppuration will occur or not, and it would be unwise to spread the infection in any way.

The breast pump is valuable in evacuating the breast to its fullest extent without in any way interfering with infection if already present. In some rare cases the local condition will discharge via the nipple, even after onset of suppuration.

The use of anti-bacterial measures has often been emphasized in the literature. Before weaning, Benians[1] employed *Sta. aureus* vaccines and found them to be valuable as supplements to evacuation of the breast. Mourgue-Molines[6] employed injections of vaccines together with poultices of 'bouillon vaccin'. Hamm,[15] of Strasbourg, basing his work on that of Besredka,[16] poulticed the skin of the lactating mother with antitoxin as a prophylactic. Whether all these measures are of any value we cannot decide in the Out-patient Department of a large hospital. As a criticism we may say that no attempts at control experiments appear to have been made by these authors.

Unfortunately we have no controls either, and can only state that for ordinary

purposes of hospital work these anti-bacterial measures appear to be unnecessary. After all, engorgement is not necessarily followed by suppuration. Relief of engorgement cannot be obtained by anything but evacuation of the breast. It is possible that where attempts at evacuation fail, the course of infection is less fulminating where the patient's resistance has been raised. However, the whole process of engorgement, if succeeded by suppuration, is such a rapid one that it is doubtful whether the measures of inoculation described will have had time to produce their maximum effect.

We have obtained excellent results in engorgement with the sole use of the simple measures enunciated above.

CLINICAL COURSE OF BREAST ABSCESS

There are various types of breast abscess met with in clinical experience. The superficial or subareolar type of abscess follows a direct infection of subareolar tissue and need not necessarily bear any relation to lactation. The deep abscess is that seen most commonly in direct connection with lactation. The type varies: (*a*) According to the type of engorgement so commonly a precursor of the condition; (*b*) According to the virulence of the infection; (*c*) According to the resistance of the mother to the infection concerned.

As we see it, the course of events is as follows:—

1. Engorgement of the breast is the primary factor in the production of breast abscess.

2. The retained secretion acts as a suitable nidus for organisms which have constant access to the breast, if they are not already present in the ducts.

3. The milky engorgement has already seriously affected the resistance of the functioning glandular tissue, and the peri-alveolar and peri-ductular structures become rapidly involved in the suppurative process.

4. According to the type of engorgement—namely, that generalized throughout the breast, or that localized to one lobe—infection starts in several portions of the breast separately, or in only one place. The progress of infection now depends on the virulence of the infection and the resistance of the patient.

5. Under good conditions the generalized type will localize and multiple well-localized abscesses will result. Under bad conditions, particularly those seen amongst out-patients in London, the whole breast slowly becomes involved in a cellulitis in which localization is poor. Successive areas become involved and break down, and unless active treatment is pursued the whole breast becomes disorganized and ultimately functionless.

6. Where one lobe only remains engorged the infection usually appears to remain localized, and if adequate treatment is adopted, then only that portion of the breast will be affected functionally. If treatment is delayed, then the interlobar septum becomes first invaded, then destroyed, and infection spreads.

7. In any case, should treatment of the condition be inadequate, then either the abscess will burst through the skin and, if still untreated, give rise to a chronic sinus which communicates with disorganized breast tissue, or else the whole breast will become one huge abscess and all functioning elements will be destroyed.

From the above it will follow that one would expect to encounter the following types of abscess:—

1. A diffuse reaction of a cellulitic nature with progressive involvement of tissue, and formation of numerous soft areas containing pus. Constitutional symptoms are probably marked.

2. A breast the seat of one or more localized abscesses with intervening tissue that appears to be fairly normal. The degree of toxæmia varies from case to case. There may be very marked constitutional reaction or practically none at all.

3. A breast that on examination appears to be one huge abscess.

4. A chronic sinus in a breast that has been for some time the seat of suppuration following inadequate treatment.

DETAILS OF TREATMENT

Incision and Drainage.—At one time we believed that there was only one type of treatment suitable for breast abscess—namely, incision and drainage. In consequence we treated our first 42 cases by this means. The procedure was as follows :—

1. When she was in a very toxic condition, we admitted the patient; otherwise we saw her daily in the Casualty Department.

2. If there was definite fluctuation present in the area concerned, we resorted immediately to surgical treatment. On the other hand, if there was a lump present, with redness, tenderness, and indefinite fluctuation, we allowed twenty-four hours with breast support and careful use of the breast pump to see whether by any chance the condition would settle. (We kept notes of five cases of this type that settled, which are not part of the series.)

3. Once incision had been decided upon, the procedure employed was as follows :—

N_2O anæsthesia was employed in all cases. If the abscess was situated above the nipple, an incision radial to the nipple was made at the point of maximum tenderness, and the gloved finger was inserted through this to explore the abscess. All septa were broken down, and the abscess rendered as far as possible unilocular. A counter-incision was then planned and made at the most dependent point—usually at the lower and outer corner of the breast. A long piece of corrugated rubber drain was then inserted well up into the abscess through the lower incision, and kept in position with one salmon-gut stitch, and a dry dressing applied. Where the abscess was situated in the lower quadrant of the breast, only one incision was usually necessary and the abscess was explored from the lower and outer corner of the breast, a drain being then inserted as above.

4. *After-treatment.*—Dry dressings were employed exclusively until the drain was removed, and the wounds dressed twice a day. The co-operation of a district nurse in the first few days is invaluable. The drain is maintained in position until the discharge definitely diminishes and all signs of acute inflammation have disappeared. This may mean maintaining drainage for any length of time up to one week or ten days. It always pays to leave the drain in a long time, and nothing is to be lost by doing so. (This appears to be contrary to the experience of Moon and Gilbert.[18])

Mr. Mitchiner, to whom we have both been House Surgeons, has always condemned hot baths and syringing of the wound as a routine treatment. They

appear to maintain a continuation of the inflammatory process, with pocketing of pus, and in some cases in which they were being employed, a resort to dry dressings instead has resulted in a fall of temperature to normal, and a rapid cessation of local activity.

Two questions arise: (1) When is weaning to be resorted to? and (2) Should the counter-incision be made through normal breast tissue?

There appears to be no need for sudden and complete weaning of the baby in every case. It was reserved, in our series, for those cases in which the mother was extremely toxic (notably those cases that were too ill for treatment as out-patients) and those cases that did not appear to be settling after incision as out-patients.

Should the baby be weaned, it is essential to continue evacuating the breast with a breast pump. Ordinarily, we took the baby off the breast concerned and gave it supplementary feeds. As the incisions healed, and all acute inflammation subsided, then it was possible to put the baby back again on the breast.

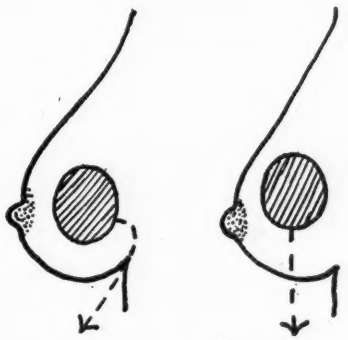

FIG. 444.—Breast abscess. Dependent drainage obtained only by cutting through normal mammary tissue.

With regard to the second question, it has long been stated that an incision down to an abscess through normal breast tissue leads to spread of infection to the normal tissue concerned. Shield[14] enunciated the principle that the counter-incision must be made through the inframammary sulcus, with the double advantage that the incision remains concealed and that the normal tissue remains free of superadded infection.

In actual fact the number of cases in which dependent drainage can be established in this way is very small indeed. If practicable, the idea is excellent. As shown in *Fig.* 444, dependent drainage can only be established by cutting through normal mammary tissue. In our experience there need be no hesitation in doing this should the necessity arise. We have never seen spread of infection in this manner. If any spread occurs it does so at the upper portion of the breast, owing to lack of dependent drainage.

FIG. 445.—Incision above nipple. No dependent drainage.

RESULTS.—We treated in this way 7 abscesses in non-lacating breasts and 35 in lactating breasts.

Non-lactating Breasts.—In the non-lactating cases only 1 gave rise to any difficulty.

Case 1.—Mrs. A. B., of Battersea, aged 22 years, had noticed a lump in the upper portion of the right breast, getting larger and more painful for fifteen days. On examination the abscess was large and situated above the nipple. A somewhat bold incision had been made into a large abscess, with no attempt at dependent counter-drainage (*Fig.* 445).

Considerable hæmorrhage occurred, and the patient had to be admitted for ligature of the bleeding point. Several further incisions were made for pocketing, and the patient was treated for a fortnight with hot baths and the continual applications of hot fomentations. The case was transferred to the Septic Block after fifteen days' treatment. There was continued pyrexia. The breast was soggy and red, with some discharge from various partially healed incisions. All baths were immediately stopped and dry dressings applied instead. The temperature fell to normal in one day and the condition completely settled (*Fig.* 446).

It may well be said that the improvement at this juncture was a coincidence—it possibly was. Nevertheless the case is one for reflection and illustrates well the points to be avoided in the treatment of breast abscess by incision.

The remaining 6 cases settled and were discharged in 8, 7, 10, 3, 6, and 12 days respectively, an average period of treatment of just over a week. These figures bear out our previous statement as to the prognosis in the non-lactating breast.

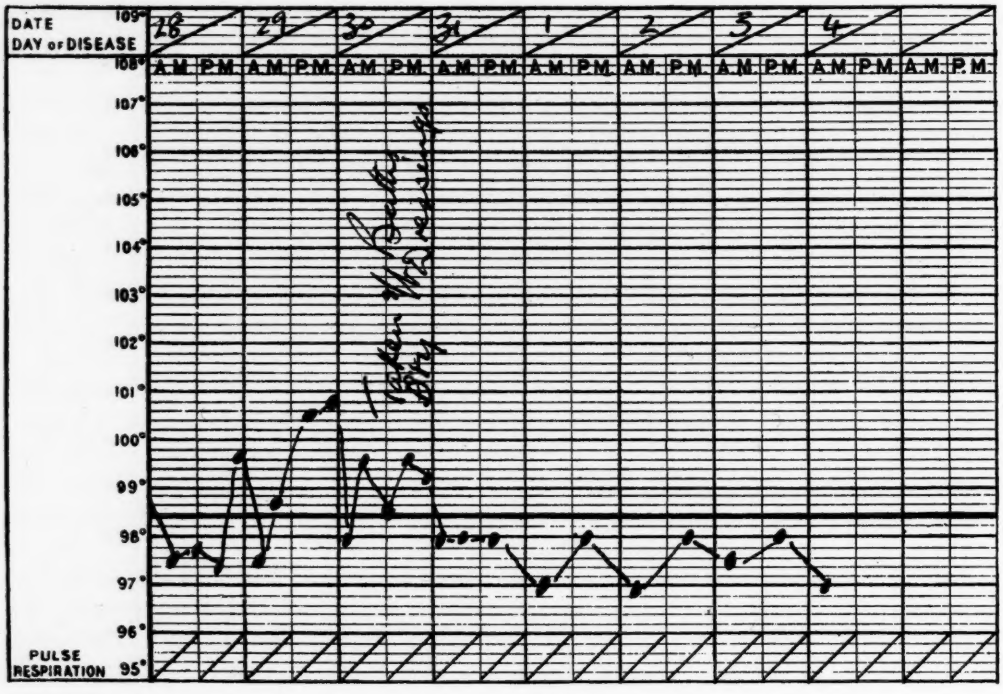

FIG. 446.—*Case* 1. Temperature chart showing effect of discontinuing baths and applying dry dressings instead.

Lactating Breast.—Very different is the prognosis in the lactating breast. Of 35 cases, as many as 11 gave difficulty and required further treatment. There are many factors that might be held responsible for these difficulties. Possibly: (1) The time of onset of infection during the lactation period; (2) The possibility of some particular type of infection associated with a cracked nipple; (3) The abscess occurring in a primipara as opposed to that in a multipara; (4) The abscess that has been neglected until it is allowed to fill the breast or discharge of its own accord; (5) The difficulty of obtaining dependent drainage with abscesses in the upper quadrant. We have examined all these possibilities and come to the conclusion that there are two main factors that complicate successful treatment when this is carried out on the principles mentioned above: (1) The stage which the abscess has reached when first seen by the surgeon concerned. (There is no fixed time factor here, as the condition depends on the subsidiary factors such as the virulence of the infection and the patient's general condition.) (2) The type of infection present. These points are best explained with an analysis of our cases

treated by incision. We have referred above to the various cases met with according to the nature and course of the infection.

1. First, we have the type of single localized abscess occupying one lobe of the breast: 26 of our cases fall into this category, only 7 of which gave any trouble at all. Five of these had to be incised a second time; 1 settled on weaning of the infant; and 1 case developed acute mania of the puerperal type, although there was no difficulty in the treatment of the local condition. (This patient had to be transferred to a mental hospital.)

Of the 5 cases in which re-incision had to be resorted to: in 1 case the drain was removed in the first instance for primary hæmorrhage, and pocketing occurred in consequence; in 1 case at the second operation no pus was found and the case would probably have settled without this second incision; and 1 case flared up six weeks after discharge, apparently on resumption of normal breast feeding. Of the remaining 2 cases that failed to settle, mistaken treatment appeared to be the only factor responsible for failure; 1 case had quite settled and was sent to a convalescent home, where the pernicious practice of 'plugging the abscess' was resorted to, and in 1 case drainage in the first place was neither adequate nor sufficiently dependent.

2. Secondly, we have the type of multiple localized abscesses. One case of this type was treated by us with incision and only one abscess was drained. At the second operation undertaken for pocketing, an abscess cavity, quite separate from the first, was found, and no further trouble was experienced.

3. Thirdly, there is the most dangerous type of mammary infection—namely, the cellulitis that slowly spreads throughout the breast and breaks down at numerous places. We encountered two of these unfortunate cases:—

Case 2.—Mrs. A. P., aged 31, a primipara, had her confinement on Jan. 25, 1934, in St. Thomas's Hospital. Both nipples cracked soon afterwards and the baby was weaned. Whether the breast was insufficiently emptied, or whether weaning was performed too late, it cannot be decided, but masses appeared in each breast associated with a pyrexia—on the left side on Feb. 8, and on the right on Feb. 12. She was first seen by us on Feb. 12 and was then in an extremely toxic condition, with a hard mass on each side, and was admitted to the Septic Block. Both breasts were repeatedly incised (six times in all). Infected areas were broken down with the finger, and the whole drained at the most dependent point. Following each operation there was a 'flare up' with a high temperature and rapid pulse, followed by all the signs of settling down. Then the temperature would again begin to swing, and fluctuant areas would appear. The whole condition ultimately subsided completely, after two months of active treatment.

Case 3.—Mrs. A. T. Her first baby was born on April 5, 1934. Both nipples were sore and nipple shields were employed. A lump appeared in the right breast, and was incised on April 20 above the nipple, but dependent drainage was not assured.

The patient was treated elsewhere in hospital for a month and was first seen by us on May 22. At this time the whole breast was involved and there were five separate areas of fluctuation arranged round the nipple. The remainder of the breast was hard and tender. The patient was forthwith admitted to the Septic Block. Incisions were made at each of the fluctuant areas, intermediate tissue was soft and easily broken down with the finger, and the whole cavity well drained. The condition settled immediately, drains were removed on the eighth day, and the patient discharged nearly healed a week later.

It will be stated that this cellulitic condition only arises as a result of mistaken treatment in the first instance. We can but state that such a condition occurs not infrequently and appears to us to be a definite clinical entity in itself. In our view mistaken treatment is most commonly followed by the next types.

4. This type of case is that of the condition that has been left until the whole breast is one large abscess cavity, and is a condition that should surely be avoidable. Three of our cases came into this category of advanced breast abscess. In spite of adequate drainage, two of these had to be incised again. One case was pocketing in the upper quadrant of the breast, and settled with a second incision in the infra-mammary sulcus, and one case pocketed below but also settled with one further incision.

5. The last group of cases is that in which the patient has been allowed to remain untreated until the abscess has burst and a sinus has formed. Three of these cases came under our care. All gave difficulty. Two had to be re-incised, and one settled with general ultra-violet ray treatment only.

The cases that give difficulty are thus firstly that type of infection that produces a cellulitic reaction, and secondly, the type of case that is left for a long time before surgical treatment is attempted. The multiple abscesses should not really be the cause of trouble. Careful search should be made to exclude the presence of a separate small abscess.

The prognosis is good in cases in which, when first seen, the infection is localized to one lobe, and adequate surgical treatment is promptly undertaken.

The results of incision were sufficiently poor to drive us to explore the literature for further and possibly more satisfactory methods of treatment. During this transition period we were able to treat a case by Hamilton Bailey's[3] method, with decided success. Although the method is doubtless excellent for in-patient treatment, it appeared to be rather complex for use in a crowded Out-patient Department, and repeated visits by a district nurse became necessary.

Aspiration.—

a. Treatment with 'Bouillon Vaccin'.—We therefore turned to the method of treatment advocated by Professor Riche and Dr. Mourgue-Molines.[7] This method has the following advantages: (1) General anæsthesia becomes unnecessary; (2) Scarring is not produced; (3) There is no interference with function of the normal breast tissue; (4) The post-operative period is absolutely painless, and close attention to the patient becomes unnecessary, as there are no dressings to be performed; (5) Most important of all, the authors describe a case of the cellulitic type in which treatment completely localized the infection.

The method employed by these workers consists in the aspiration of the abscess and the employment of a bouillon vaccin produced by a firm in Paris, which consists of a filtrate of cultures of staphylococci, etc., according to the type required. The procedure is briefly as follows: The skin over the most tender point in the abscess is infiltrated with novocain. A wide-bore needle is then inserted into the abscess cavity and pus is evacuated. The cavity is washed out with bouillon vaccin, and finally a quantity of the latter—equal to half the amount of pus removed—is left in it.

A profound reaction should ensue. The temperature and pulse should rise, the abscess should become tense and possibly painful over night. The next day, however, the condition should appear more localized, the pain should disappear, together with the more dramatic constitutional symptoms. On the third day, if no further improvement is taking place, the aspiration is repeated. According to the authors, several applications are necessary until the condition has completely

settled. They have obtained quite unusual results, which led us to follow their example, and we were fortunate in obtaining permission from the Ministry of Health to import samples of bouillon vaccin from the firm in Paris, to whom Professor Riche had very kindly sent a note on our behalf.

The ' No. 31 ', which was apparently the most suitable of the various bouillons vaccins on the market, formed only a small part of the consignment. Consequently we very rapidly exhausted our small stock, and were forced to employ the other types—designed for such varying diseases as metritis and furunculosis. These acted as excellent controls, and actually produced better results than the ' No. 31 '.

Altogether we had a sufficient supply of bouillon vaccin to treat 21 cases. Two of these cases were in non-lactating patients (in one we used ' No. 31 ' and in the other ' No. 41 '). They both did well, and could be discharged in less than a week each as there was no wound to heal. Of the remaining 19 cases, one must be excluded from the series as we have no record after her first attendance and cannot trace her for follow-up purposes. This leaves us with 18 cases, of which 4 had ultimately to be incised—all lactating patients. One patient failed to report and had to be sent for. On her ultimate return, the condition had progressed, and not understanding very well the scope of aspiration, we decided to resort to incision. One patient reported on the evening of her first attendance with marked constitutional disturbance, a temperature of 102° and a pulse of 120. She was admitted to the Septic Block, the abscess was incised the next day, and settled down quite well without further intervention. We believe that she would have done quite well with further aspiration. Two patients failed to settle with repeated aspirations and even weaning of the infant. They were admitted to the Septic Block and incised and even then failed to settle immediately.

All these four cases were treated with ' No. 31 ', apparently the ideal bouillon vaccin. Our technique was poor, as we had no experience of our subject, and it is possible that, had we treated these cases later in our series, instead of amongst our first 8 or so patients, we would have obtained better results. We did, however, appreciate that there was no specific property to justify the faith reposed in ' No. 31 ' by Riche and Mourgue-Molines. Our control cases, performed with non-specific bouillons vaccins (Nos. 10, 24, 41) had done better. This appeared to confirm the findings of others. Laffont, Fulconis, and Ricard[18] were able to cure 5 cases by aspiration and injection of peptone solution. As the bouillons vaccins are all made up with peptone, it would appear that their effect is possibly one of protein shock.

In one case we decided to do a control with normal saline solution in a patient with bilateral abscesses. We washed out the cavity on one side with saline only, and on the other with bouillon vaccine ' No. 31 '. It required four injections of saline to settle one side, whereas the other side settled with one injection of bouillon vaccin ' No. 31 '. At any rate, we can say that in this case the action of the bouillons vaccins of Riche and Mourgue-Molines was not purely a mechanical one.

We soon ran out of our materials provided through the courtesy of Professor Riche. In order to avoid a prolonged correspondence with the Ministry of Health and heavy customs duties, we resorted to Dakin's solution. In doing so we were on safe ground.

b. Treatment with Dakin's Solution.—Dr. Alejandro Pavlovsky and others have successfully treated breast abscesses with antiseptic solutions. Hobbs[10] has

obtained good results with incision and irrigation with Dakin's solution. Hamilton Bailey[3] employs it in the treatment he describes.

Our method was essentially as described above, without the production of quite the same constitutional reaction in the patient. We were able thus to treat 7 cases; 2 of these in non-lactating and 5 in lactating patients. Of the former, 1 gave no trouble at all, and the other settled very well on ensuring extra food, and applications of general ultra-violet light.

Of the 5 lactating cases, all did well with one exception. This was a patient in the ward attached to the Venereal Department, who developed a breast abscess on the right side early in the puerperium. The temperature remained exasperatingly high, in spite of several aspirations, and careful washouts with Dakin's solution. There were practically no signs in the other breast except slight tenderness and possibly a fullness in the upper and outer quadrant. Finally we explored the other breast with a needle, evacuated somewhat to our surprise 30 or 40 c.c. of pus, and washed out the cavity. The temperature still did not fall, and under N_2O anæsthesia we incised an enormous abscess and drained it from the most dependent point. The temperature then settled at once, and although the breast discharged over a long period, the wound was healed within one month of commencement of the trouble.

It appears impossible at first for the person who is wont to incise breast abscesses to understand that adequate drainage can possibly be maintained by repeated aspirations. We started this treatment under strong criticism, and with serious doubts in our minds as to the efficiency of methods described by the various authors we read.

J. W. S. H. Lindahl, until recently the House Surgeon to the Septic Block, has successfully treated quite a number of cases by aspiration and washing out with Dakin's in the same way.

Technique.—The exact technique employed in treating a case by aspiration is not of particular importance. Anybody who tries this method will sooner or later employ a technique of his own according to the instruments and facilities at his disposal. That which we describe here is one which we have developed ourselves, and which we trust may prove of value as a starting point to any persons interested in this method. When it was decided to aspirate the abscess, the baby was immediately taken off the breast concerned and given complementary feeds when necessary. Care was then taken that all milk forming in the affected breast should be removed at regular intervals with the aid of the breast pump. The syringe employed is of a capacity of about 25 c.c., to which may be attached a needle of wide bore. We have employed needles of a diameter of 2·5 mm., and like to have at least two, and preferably three, of these ready, sterilized for use with every case. The best type of syringe is that in which the needle is attached by a 'Luer' fitting, the 'record' attachment being too narrow to allow successful passage of sloughs into the barrel.

The breast is cleansed with ether and the surrounding area packed off with sterile towels. The point at which aspiration is being performed must then be chosen. This point is that at which fluctuation is present. If seen early the abscess should be aspirated at the point of maximum tenderness. The area chosen is then infiltrated with 2 per cent novocain down to the abscess cavity, which is easily recognized owing to the facility with which the anæsthetic enters. The plunger of

the 'record' syringe is then withdrawn and the position of the needle is often confirmed by the withdrawal of pus.

A wide-bore aspiration needle is then inserted through the anæsthetized area into the abscess cavity and pus withdrawn. The needle is left in position, the pus in the syringe is emptied out, and an equal quantity of Dakin's solution diluted to half strength with sterile water is inserted again via the needle *in situ*.

Should the needle become blocked during aspiration, the syringe should be emptied again gently towards the abscess cavity, or else more Dakin's solution should be injected. It should never be necessary to remove the needle until the whole procedure is accomplished. In this way the cavity should be washed out until the fluid in the syringe is nearly clear, and at any rate until no more debris and sloughs are recognizable in the fluid removed. The abscess cavity should be left empty, and by withdrawal of sloughs it is hoped that the walls may be allowed to fall together.

After the aspiration the point of entry of the needle should be covered with the minimum dressing and the breast supported with a proper sling bandage. There is no need for any further local treatment now until the patient reports for further inspection in about twenty-four hours' time.

One aspiration is often sufficient in treating the small superficial abscesses, but for the commoner case of deeper abscess, repeated aspirations are usually necessary. Here are the indications for further aspiration :—

1. *Continued Pyrexia.*—When present alone without local signs, aspiration can be deferred for a further twenty-four hours, particularly when leakage is occurring through the puncture in the breast.

2. *Pain.*—Pain in the breast concerned is immediately relieved by aspiration of the abscess cavity and is evidently merely the result of tension. With return of pain aspiration is indicated.

3. *Local Tenderness.*—If it persists, or if it returns, this is a strong indication for further interference.

We would emphasize that in the absence of these indications aspiration need not be repeated. We have found in our own experience that there is a strong temptation to repeat aspirations quite unnecessarily, and careful consideration should always be given to the case before further aspiration is attempted.

Failure to settle after two or three aspirations is not uncommon. Several considerations must now be borne in mind, and we have proceeded on the following lines :—

1. Weaning of the baby. Several of our cases have settled on complete weaning without further aspiration. In these cases, of course, it is essential that both breasts should be kept carefully emptied by means of the breast pump.

2. The general condition of the patient must be considered. Remarkable improvement will sometimes be seen when extra nourishment is provided in the form of milk, eggs, etc. In all cases we have prescribed alcohol where possible, preferably in the form of the malted liquors. General applications of ultra-violet light have appeared to be valuable in the only two of our cases in which they have been tried.

3. Cases are frequently met with in which several loculi drain into a main abscess cavity, and which continue to maintain signs of activity in the breast when the main abscess cavity appears to be quite settled.

When first seen these cases may present two or more areas of tenderness, widely separated from each other. It is as well, then, to aspirate each cavity separately with a needle inserted at these points and then with the needles *in situ* to wash the Dakin's solution to and fro between the separate loculi.

Should the area of tenderness, or presence of fluctuation, suggest that some loculus is maintaining the toxæmia, then it is essential that these areas be explored and carefully washed out. In one of our cases we found as many as four different loculi forming separate abscesses in close contact with each other. Fortunately the condition was recognized early and the case settled well without the necessity for incision.

Finally, if in spite of these considerations, the case fails to settle after repeated aspirations, resort must be had to incision, which we carried out in our series on the lines indicated in the first section of this article. As we increased our experience of aspiration, we found that the number of cases which failed to settle with this treatment very greatly decreased in number. Nearly all our failures occurred amongst our early cases. It should also be stated that all our cases of aspiration were treated as out-patients.

CONCLUSIONS

It has been stated elsewhere[19] that variations in local treatment by different surgeons is of no significance. We cannot agree with this statement, and have found that increase of experience in the treatment of intramammary abscess is accompanied by improved results. Consequently we have entered into considerable technical detail in this series of cases, some of which we have treated with incision and some with aspiration.

We have described a method of aspirating breast abscesses and washing them out with Dakin's solution. Abroad, certain ' bouillons vaccins ' have been employed with conspicuous success. To import these it is necessary to obtain special permission of the Ministry of Health and to meet customs obligations. Dakin's solution has been found to give good results, and is of easy access to any practitioner in this country.

It now becomes necessary to review the whole series of cases and to attempt some impartial judgement between the merits of incision and those of aspiration.

1. The aspiration of an abscess is a procedure that can be performed single-handed without skilled assistance. The anæsthetic employed is a local one, there is no wound to be dressed afterwards and the co-operation of a district nurse becomes unnecessary. Scarring is reduced to a minimum, and consequently the ultimate cosmetic and functional result is of the best. Aspiration is therefore to be preferred in those cases of suppuration occurring in the non-lactating breast and in those cases in which, during lactation, the abscess is relatively localized and confined to one lobe.

2. With the very large abscess and neglected breast, incision immediately relieves an already prolonged toxæmia and drains a large cavity more satisfactorily than can aspiration.

3. With the diffuse, cellulitic type of infection the prognosis is poor, however the condition be treated. Incision is probably to be preferred, in that by this means the local condition can be thoroughly explored with the finger and infected areas broken down into one large abscess cavity.

4. Should any doubt be entertained as to the treatment necessary with any particular case, we advise the use of aspiration. At the worst incision can always be resorted to with failure to settle after several aspirations, and only time is lost.

In conclusion we should like to record our indebtedness to many who have helped us directly and indirectly with the preparation of this article: Mr. P. H. Mitchiner, to whom we were both House Surgeons in the Septic Block of St. Thomas's Hospital, for the permission to use his cases and for his excellent advice and friendly criticism; Professor Riche, who very graciously gave us an introduction to the Maison Gremy of Paris, so that we could obtain a supply of bouillon vaccin; Dr. Doyne Bell for his help with difficulties encountered in the writing of this article; Drs. J. P. Hedley, R. Jewesbury, J. Wyatt, P. A. Clements, and J. Wrigley, for permission to quote cases of theirs which we have treated in this series; and Mr. J. W. S. H. Lindahl for his help in the correction of proofs. Finally, we would particularly thank Miss Annie Beal and her staff in the Casualty Department for their untiring support, and also Miss Routh, sister-in-charge of the Septic Block, whose knowledge of the nursing of septic cases must be unrivalled in this country.

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VISITS TO SURGICAL CLINICS AT HOME AND ABROAD

MR. H. B. DEVINE AT ST. VINCENT'S HOSPITAL, MELBOURNE

DURING the recent visit of the British Medical Association to Melbourne on the occasion of their Annual Meeting we had the opportunity of seeing the work at the hospitals, both in the city and in the outlying seaside annexes. One is conscious of the fact that the rush of meetings and festivities does not afford the opportunity for quiet observation that one would wish for, nor does it allow for an adequate appreciation or description of the hospitals and surgical clinics of a great city. But, on the other hand, one was so much impressed by the excellence of the work, that one cannot do less than give a brief description of the clinics of one of the leading surgeons who is so largely responsible for the high surgical standards in the capital city of Victoria.

Melbourne, a spacious city of over a million inhabitants, has four main hospitals, the Melbourne, the Alfred, the St. Vincent's, and the Children's. At present there is no close association between these hospitals or between them and the University, each hospital taking a certain number of students, who are examined for their degree at the University. Nor is there a Professor of Surgery at Melbourne, the senior surgeons of the three general hospitals acting as lecturers and examiners for the University.

The St. Vincent's Hospital of 250 beds has recently been rebuilt and represents the last word in up-to-date hospital architecture. The institution is a 'voluntary' hospital, but the patients pay towards the cost of their maintenance and there is an ample provision of wards for private patients.

On Tuesday, Sept. 10, Mr. Devine operated before a large number of spectators, including many members of the British Medical Association.

1. Gall-stones: Cholecystectomy (2.30 to 3.15 p.m.).—Woman, aged 61, high blood-pressure, enlarged liver. Anæsthesia by ether. Mid-line incision 6 in. long. Edges of incision protected by sponge cloths and drawn aside by retractors held on a square metal frame (*Fig.* 447). (The use of this frame retractor plays a prominent part in all Devine's abdominal work. Various types and shapes of retractor can be adjusted to any of the four sides of the square. It gives a splendid view of the abdominal cavity and makes it unnecessary to touch or handle any of the viscera.) The gall-bladder full of stones was well exposed, whilst the liver and intestines were packed away and held by covered retractors. The cystic duct and artery were exposed and ligatured and the gall-bladder was removed with a few touches of the scissors. The abdominal wound was closed in layers with catgut.

2. Colostomy for Inoperable Growth of Colon (3.30 to 4 p.m.).—Man of 50. Typical symptoms. Growth demonstrated by X rays to be in the splenic flexure. Vertical incision over the right rectus. There was a good deal of free

FIG. 447.—Mr. Devine operating. The photograph also shows the square metal frame retractor used for abdominal surgery.

fluid and a sucker was inserted into the lower angle of the wound. The hand, well smeared with vaseline, was inserted for exploration. The growth was felt to be inoperable and therefore a colostomy was decided upon. The hepatic flexure

FIG. 448.—Mr. Devine conducting a students' clinic.

was mobilized and brought to the surface. A large tube was passed through the mesocolon to hold the gut on the surface. The two limbs of the loop were sewn together proximal to this. Two Kocher's forceps were applied across the gut,

which was divided by a diathermy knife. A skin-flap was then fashioned from the outer part of the abdominal incision and turned up so as to lie between the two divided ends of the colon. This skin was sewn in place and was made to surround each stoma tightly. Both clamps were left on; that on the proximal end was to be removed in twenty-four hours, whilst that on the distal was left till it came off.

3. Carcinoma of Male Breast: Radical Removal (4.15 to 5 p.m.).—Man, aged 63. Nodule medial to nipple on left breast. General anæsthesia. Skin incisions first marked out by light cuts. Wide area of skin removed. Diathermy not used because of the danger to vein. Dissection done entirely by the scissors. Axilla cleared first. The breast and axillary contents having been

FIG. 449.—The Royal Australasian College of Surgeons, Melbourne.

removed, the vessels were ligatured with silk. The large raw area left was closed first by turning down a flap of skin from the chest and then by a number of Thiersch grafts taken from the right thigh. These were covered by tin-foil over which pieces of rubber sponge were placed to maintain pressure.

The wards of St. Vincent's Hospital are large and well lit. The nursing is by the sisters of St. Vincent de Paul. Ward rounds, well attended by students, give great scope for Devine's teaching faculty (*Fig.* 448). He is on the most friendly terms with his dressers and house surgeons, and obviously has impressed them all with a great sense of loyalty and devotion.

It would not be fair to close this very brief and inadequate account of Devine's work without referring to two other directions in which his energies have made

themselves felt in the surgical world of Australia. The first of these is the Royal Australasian College of Surgeons (*Fig.* 449), opened at the beginning of this year by Sir Holbert Waring. Both Hamilton Russell and Sir Richard Stawell were active in initiating the movement for the foundation of the College. But no one has done more in an unobtrusive way than Devine for securing the ground and the funds, and, above all, reconciling conflicting views and inspiring the ideal which underlies this College. It is a dignified red brick structure splendidly situated in a wide open space quite near to the Parliament House. Dr. Wade, of Sidney, is the President of the College, and Mr. Allen Newton shares with Mr. Devine the greater part of the work of planning and organization.

The second great piece of constructive surgical work which stands to Devine's credit is the foundation and editing of the *Australasian Journal of Surgery*. It is a great and ambitious undertaking for a country of the population of Australia and New Zealand, and in its successful conduct we wish H. B. Devine every success.

EXPERIMENTAL SURGERY

THE INFLUENCE OF ŒSTROGENIC COMPOUNDS IN CAUSING HERNIA AND DESCENT OF THE TESTIS IN MICE

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THE causation of scrotal hernia in mice by the administration of œstrone (œstrin) was described by the author in a previous number of this JOURNAL.[1] Since that article was written, further observations have been made in this branch of pathology which are perhaps worth recording.

1. Scrotal Hernia not Found in Mice apart from the Administration of Œstrogens.—A matter of importance was to learn how often, if at all, scrotal hernia might develop in mice which had not been submitted to any œstrogenic compound. For this purpose 4013 male mice of all ages were examined. Some of them were untreated mice, others had been grafted with tumours or subjected to various tests in connection with the production of cancer; none of them had received applications of any œstrogen. No instance of scrotal hernia was discovered in the whole of this series. The appearance of scrotal hernias in mice after the administration of an œstrogenic compound may therefore be safely attributed, it seems, to the action of that compound.

2. Substances which Cause Hernia in Mice.—In the course of our experiments eight different œstrogenic compounds, chemically pure, have been found to induce hernia. Their relative capacities for causing hernia vary, and seem to correspond to some extent, though not entirely, with their œstrogenic potency. More precise statements cannot be based on the experiments now in consideration; the great majority were designed for another purpose than the study of hernia formation and consequently did not include a regard for all the details which would be necessary for an exact study of the comparative potencies of different œstrogens to cause hernia. The compounds found to be capable of inducing scrotal hernia in mice are œstrone (œstrin), œstrone methyl ether (ketomethoxyœstrin), œstradiol (dihydroxyœstrin), œstriol (trihydroxyœstrin), equilin, equilenin, 9 : 10-dihydroxy-9 : 10-di-*n*-propyl-9 : 10-dihydro-1 : 2 : 5 : 6-dibenzanthracene (Cook, Dodds, Hewett, and Lawson[2]), and a similar compound with butyl substituted for propyl (Cook[3]) (*Fig.* 450).

Among compounds which, having some similarity of structure to the œstrogens, are not œstrogenic and do not cause hernia, are androsterone (male sex hormone), progesterone (hormone of the corpus luteum), pregnandiol, and deoxycholic acid. The formulæ of these are given for comparison with those above (*Fig.* 451).

3. Additional Observations on the Production of Hernia by Œstrone.—Since the previous paper on hernia in mice was written, further observations have been made with œstrone. A hundred more male mice have been treated with bi-weekly applications of œstrone (0·01 per cent in benzene) to the skin of the back: 22 early deaths occurred in this group, and among the 78 survivors hernia occurred in 47.

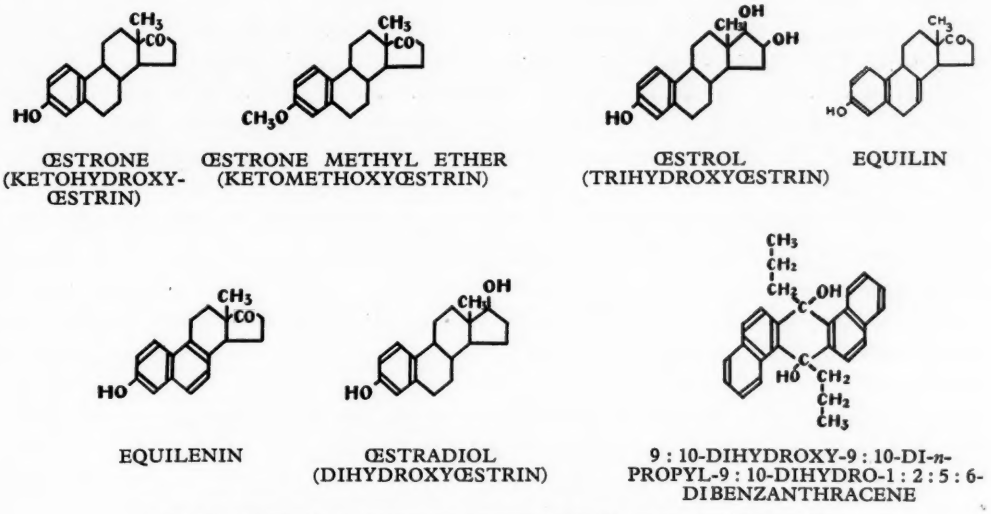

FIG. 450.—Formulæ of œstrogenic compounds which cause hernias in mice.

a. Effect of Non-descent of the Testis.—An early question to arise was why hernia should appear in some mice and not in others treated in exactly the same way with œstrone. It was noticed that hernia occurred most readily in those mice whose testes were fully developed and lying well down in the scrotum when the applications of œstrone were begun. Most of our experiments were carried out on young mice freshly arrived from the dealer, and said to be between 5 and

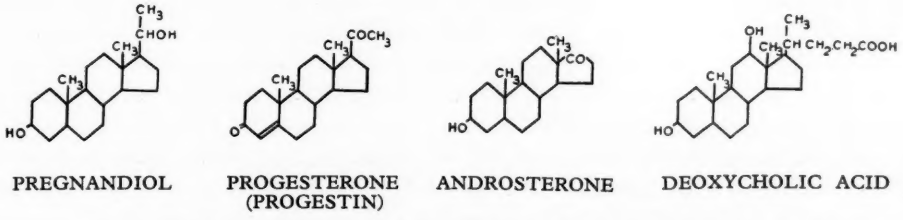

FIG. 451.—Formulæ of compounds allied to those of *Fig.* 450, but which do not cause hernias or artificial œstrus in mice.

6 weeks old. Among 1410 of these mice examined consecutively, the testes were undescended in 427. When such immature mice with undescended testes are submitted to regular bi-weekly applications of œstrone, some of the testes come down into the scrotum, but many remain undescended so long as the treatment with œstrone is maintained; and as long as the testes remain in the abdomen no hernia occurs. Two facts seem to have emerged: (i) The presence of a more or less mature testis is necessary for the formation of scrotal hernia under the influence

of œstrone; (ii) Œstrone may prevent descent of the testis, which in this event fails to become mature, as shown by its small dimensions and by the absence of spermatogenesis.

Hernia does not invariably follow the application of œstrone even when the testes descend into the scrotum (*see Table II*). The explanation of this must await an opportunity to examine the testes at the time when hernias first appear in a group of mice submitted to the necessary œstrogens.

b. Effects of Artificial Cryptorchidism.—Thirty-eight mice whose testes were fully descended were rendered cryptorchid by dividing the gubernaculum on each side and displacing the testes into the abdomen. In 18 of the mice the operation was performed by means of a laparotomy wound so as to avoid injury to the scrotum, and in 20 the exposure was made through the scrotum. Of the 38 mice treated in this way, 28 were given applications of œstrone, and 10 were kept as controls. Hernias subsequently occurred in 15 of the mice treated with œstrone, and no hernia occurred among the controls (*Table I*).

Table I.—Effects of Artificial Cryptorchidism Combined with Applications of Œstrone

Number of Mice	Site of Operation	Subsequent Treatment	Mice with Hernia
18	Abdomen	Œstrone	9
10	Scrotum	Œstrone	6
10	Scrotum	None	0

Sixty-eight days after the operation on these mice there were 34 survivors, of which 25 had been treated meanwhile with œstrone, and 9 had not been given œstrone. Among those treated with œstrone descent of the right testis had occurred in 19 and descent of the left in 18. Among the 9 which had received no œstrone the right testis had descended in 8 and the left in 6. In most and perhaps in all the instances in which retention occurred it was attributable to fixation of the testis in the abdomen by post-operative adhesions.

In some of the mice in this experiment a bilateral hernia was found, although one of the testes had been retained in the abdomen by post-operative adhesions. In one instance the left testis had failed to descend because of adhesions, and the right testis had descended into the left scrotum. In another mouse both testes had come down into the left scrotal pouch. In both of these mice bilateral hernias were present. These observations show that a gubernaculum is unnecessary for the descent of the testis, though it is indispensable as a guide to a correct disposal of the testis in the scrotum. Further, although a well-developed testis is necessary for the production of a scrotal hernia by œstrone, that testis need not itself be in the scrotum at the time of formation of the hernia.

c. Effect of Unilateral Castration.—To elucidate the matter, the right testis was excised from 10 mice, and applications of œstrone were thereafter made to them and to 10 intact controls. Bilateral hernias appeared in 4 of the survivors of the mice with a single testis and in 6 among the 10 which had not been castrated (*Table II*).

Table II.—Effects of Applying Œstrone to Mice Having One Testis Only

Number of Mice	Preliminary Treatment	Surviving after 65 Days	Number with Retained Testis	Mice with Bilateral Hernias
10	Unilateral castration	5	1	4
10	None	10	1	6

In each series no hernia occurred in the mouse with retained testis.

d. Effect of Bilateral Castration.—The testes were removed from 30 mice, which were thereafter treated with applications of œstrone till their death. In none of these mice did any hernia appear.

e. Effect of Vitamin Deficiency.—The healthy development of seminal epithelium is dependent upon adequate supplies of vitamins in the diet. Vitamin E is of particular importance, a deficiency leading rapidly to degeneration of the seminal epithelium. Ten mice were placed on a diet deficient in vitamin E, and another 10 were given a diet of white bread only, and both these series were thereafter treated with œstrone in the usual way. A heavy mortality ensued. At the end of six weeks only 3 of the mice on a vitamin E deficient diet remained alive, and none showed a scrotal hernia, though the period of treatment was sufficiently long for such a development. Of the mice kept on a diet of white bread, 5 survived for three months or more, and no scrotal hernia developed (*Table III*). Among 10 control mice treated with œstrone and given a normal diet, hernias developed in 7.

Table III.—Effect of Vitamin-deficient Diets in Preventing Hernia in Mice Treated with Œstrone

Number of Mice	Diet	Surviving for 6 Weeks or More	Mice with Hernia
10	Vitamin E deficient	3	0
10	White bread only	5	0
10	Normal	9	7

In a later experiment 30 mice were treated with bi-weekly cutaneous applications of œstrone benzoate (0·01 per cent in benzene). Thirteen weeks after the commencement the results were as shown in *Table IV*.

Table IV.—Production of Hernia by Œstrone Benzoate

Number of Mice	Diet	Surviving at End of 13 Weeks	Retained Testes	Mice with Hernia
20	Normal	16	0	5
10	Vitamin E deficient	4	4	0

f. The Effects of Long-continued Applications of Œstrone.—In some cases the hernias have progressively increased so as to reach very large dimensions, and there have been several instances in which almost all the abdominal contents except parts of the liver and stomach have become transferred to the scrotum. In examples of this kind the entire spleen is sometimes found in the right scrotal pouch and sometimes in the left. Even large hernias have diminished in size on cessation of the dosage with œstrone, though they have not disappeared. Small hernias which have not long existed will become cured spontaneously if no more œstrone is given.

When considering the effects of repeated dosage with œstrone it has to be borne in mind that mice show considerable differences in the rapidity and degree with which they respond, and also that with long-continued dosage there appears to be a tendency to develop some resistance to the hormone.

DISCUSSION

In the foregoing investigation œstrone was supplied in quantities much in excess of those which occur naturally. The gross effects of the treatment on the mamma and generative organs are proofs of this. The experiments show that œstrone will bring about a scrotal hernia in the mouse only if a testis is present and is in a mature or nearly mature condition. Perhaps this may explain to some extent the descent of the testes in animals during rut, the experimentally induced scrotal hernia representing an artificial exaggeration of the natural process. Most, if not all, the changes induced by œstrone are reversible, and hernia is not an exception unless of long standing or large.

It is not easy to explain why in some instances the continued application of œstrone should lead to a progressive increase in the size of a hernia, seeing that: (1) the occurrence of hernia is associated with a well developed seminal epithelium; and (2) œstrone has a restraining effect on the seminal epithelium. Some mechanism, at present obscure, must come into play to account for these phenomena. The inhibitory effect of œstrone upon the seminal epithelium is thought to be exercised indirectly through the anterior lobe of the pituitary gland. The active phases of the seminal epithelium appear to be brought about by a hormone derived from the pituitary gland, and it is believed that œstrone inhibits the output of this hormone (Moore[4, 5]). On the other hand, prolan is thought to stimulate the pituitary to elaborate this hormone. Such reasoning provides a basis for the use of prolan in the treatment of undescended testis; though, in a tentative experiment performed by the author with Mr. F. L. Warren, three successive daily doses of 77 mouse units of a commercial preparation of prolan failed to cause descent of the testes in any one of four mice whose testes had not descended naturally. The necessity for the presence of a well-developed seminal epithelium in order that œstrone may induce a scrotal hernia may be borne in mind when the causes of non-descent of the testis in boys are under consideration. The seminal epithelium is readily damaged, not only by excess of œstrone, but by dietary deficiencies and other adverse circumstances (Mason[6]).

It has been suggested that, as œstrone may cause distension of the bladder and hydronephrosis, the hernias may be the consequence of increased mechanical pressure during efforts at micturition. Such a suggestion can be met by the

following facts: (1) Attempts to produce scrotal hernia by digital pressure on the abdomen, whether in living or freshly killed mice, have always failed. (2) The hernias induced by œstrone may appear early, having been seen exceptionally after three applications only; vesical distension is a much later consequence. (3) Castrated mice treated with œstrone, and mice whose testes are retained in the abdomen through the continued administration of œstrone, not infrequently succumb to hydronephrosis but do not have hernia. (4) Hernias were not present in two mice which died of urinary obstruction caused by scars resulting from fights.

Additional experiments are needed to elucidate the precise condition required in the testis in order that œstrone may cause hernia. Examination of the testes of a series of mice at the time that hernias are appearing is one of these. Another desirable experiment would be the unilateral castration of immature mice, to be followed by the administration of œstrone after maturity and descent of the remaining testicle have come about. The object of this would be to ascertain whether a preliminary descent of the testis is essential for the subsequent induction of scrotal hernia by œstrone. These additional experiments must await some future opportunity.

A fact that touches on the problem is that some œstrogens (equilin, œstradiol) cause a great increase of the interstitial tissue of the testis with degeneration of the tubular epithelium. These changes occur whether the testes are retained or not, and appear inimical to the development of hernia. Although either of these compounds may cause hernia in mice whose testes are already in the scrotum, they seldom caused hernia in mice whose testes had not come down at the commencement of the application. From this experience it appears that the interstitial tissue of the testis does not co-operate with the œstrogen in the induction of hernia.

One more observation may be added to the foregoing; it has reference to strangulated hernia. The aperture between the scrotal sac and the abdominal cavity in mice is not a narrow one, and strangulated hernia has not been seen. Death has occurred in two instances from intestinal obstruction, caused in one case by torsion of the cæcum within the hernial sac, and in the other by torsion of a loop of small intestine within the sac. No adhesions were present in either instance.

SUMMARY

1. Scrotal hernia rarely if ever occurs spontaneously in mice.

2. Every potent œstrogenic compound tested has induced hernias.

3. Hernia has not been induced by any non-œstrogenic compound.

4. Œstrone will induce hernia only if the testis is nearly or quite mature.

5. Œstrone tends to inhibit descent of the testis and to prevent it from maturing.

6. In immature mice œstrone may prevent descent of the testis. In these cases hernia will not appear.

7. Only one testicle is necessary for the development of bilateral hernias under the influence of œstrone.

8. Bilateral castration prevents the production of scrotal hernias by œstrone.

9. Vitamin deficiency, which damages the seminal epithelium, prevents the production of hernia by œstrone.

10. Scrotal hernias may enlarge progressively as long as the dosage with œstrone is continued.

11. Unless large or long existent the hernias disappear after cessation of the supply of œstrone.

12. The gubernaculum is not essential for the descent of the testis. It is a guide, and in its absence the testis may descend into the wrong scrotal pouch.

13. The causation of hernia by œstrone is regarded as an exaggeration of the natural process of descent of the testis.

I have great pleasure in mentioning my indebtedness to my colleagues for help and advice in carrying out this study. The chemical basis of the work has depended entirely on Professor J. W. Cook. The maintenance of mice on a diet deficient in vitamin E was kindly supervised by Dr. E. Boyland. To Dr. Girard particular thanks are due; without his generous gifts of œstrone, œstradiol, equilin, and equilenin, much of the work could not have been done. For a supply of œstriol (theelol) I am indebted to Messrs. Parke, Davis & Company, and for additional supplies of œstradiol to the Organon Laboratories.

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AN EXPERIMENTAL METHOD OF PROVIDING A COLLATERAL CIRCULATION TO THE HEART

By LAURENCE O'SHAUGHNESSY

RESEARCH LABORATORIES, ROYAL COLLEGE OF SURGEONS OF ENGLAND AND THE BUCKSTON BROWNE SURGICAL RESEARCH FARM

Of recent years the clinical importance of the collateral blood-supply to the heart has been emphasized by the work of Hudson, Moritz, and Wearn. It appears that obstruction of the main stem of a coronary artery is compensated for by collateral channels from the remainder of the coronary circulation, as well as by collaterals from the general systemic circulation, which reach the heart from the mediastinum. There are connections between the coronary system and the pericardiaco-phrenic artery and the anterior mediastinal, pericardial, bronchial, and œsophageal branches of the aorta. It is clear that in many cases this collateral circulation is inadequate, and the infarcted area in the heart wall progresses either to an acute rupture within the space of a few days or to the formation of a cardiac aneurysm, rupture of which may be delayed for an indefinite though usually limited period. In other cases ischæmia of the myocardium is followed by permanent cardiac incapacity.

The problem of increasing the collateral blood-supply may be approached in several ways. The efficiency of the collateral supply from the coronary system depends on the measures adopted at the moment of occlusion. In the experimental work of Leriche it appears that maintenance of the systemic blood-pressure is of decisive importance; the issues raised by this concept are the subject of investigations still in progress.

Various experimental methods have been adopted to increase the efficiency of the extra-cardial anastomoses. The simplest method is to promote the formation of pericardial adhesions by the application of some irritant to the endothelial lining of the sac. Beck has reported an experiment in which he removed the epicardium and so promoted the formation of adhesions between the fibrous pericardium and the surface of the heart, and his animal subsequently tolerated almost complete obliteration of the coronary arterial tree. The method described in this paper is the application of a large omental graft with its vascular pedicle in the abdomen.

The great omentum was selected as the appropriate agent for several reasons. Personal experience with the Morison operation for ascites had impressed me with the capacity of the pedicled omental graft to form new vascular attachments. In a previous paper (1933) the use of the pedicled omental graft in experimental resection of the œsophagus has been described. At a later date Ohsawa has, quite independently, recorded his successful experience with the pedicled omental graft in resection of the thoracic œsophagus in man. The method of the experiments, carried out in the cat and the dog, is as follows. Anæsthesia is induced with chloroform and maintained with oxygen and ether under positive pressure. The

left chest is entered by a long intercostal incision and the surface of the heart exposed by incision of the fibrous pericardium. An incision is made in the left leaf of the diaphragm in the direction of its fibres, and the abdominal cavity explored until the stomach and omentum are exposed. A large segment of the omentum is separated from the rest of the organ, care being taken to avoid injury to the contained vascular trunks, and the graft so obtained is attached to the surface of the heart by one or two fine sutures (*Fig.* 452). The wound in the diaphragm is

FIG. 452.—The graft in place.

closed by a single suture in such a way as to avoid pressure on the vascular pedicle of the graft. The fibrous pericardium is not sutured, and the chest wound is closed in layers.

The technique of the injection studies is based on that described by Bromer, Zschiersche, and Wearn. The injection, at a pressure of 150 mm. Hg, was made into the abdominal aorta immediately after death with indian ink diluted with an equal quantity of distilled water. The heart was removed from the chest by severing the aorta and its other vascular connections, until its only attachment to the animal was by the omental graft.

The operation was done on fourteen cats and two dogs, and although some of the animals had been previously subjected to ligature of the coronary artery, in no case was any immediate post-operative distress observed. One animal subjected to ligature of the descending branch of the left coronary artery died of acute œdema of the lung ten days after operation, and another died fourteen days after operation from obstruction due to prolapse of the small intestine into the left chest through the wound in the diaphragm. In a large series of experiments with the pedicled omental graft this was the only accident of the kind observed. In all the other specimens studied at post-mortem the omentum was firmly adherent to the margins of the opening in the diaphragm.

There follow the protocols of several experiments of special importance.

FIG. 453.—*Dog* 1. Greyhound heart with graft in position.

Dog. 1 (greyhound, aged ?).—

8.1.35.—Operation: ligature of descending branch of left coronary artery.

15.4.35.—Exercise tolerance tested on track. After completing the course the dog appeared rather more distressed than a normal animal as judged by colour of tongue and respiration rate.

16.4.35.—Operation: pedicled omental graft applied to area of old infarction in heart wall.

12.9.35.—Exercise tolerance again tested on track. Performance normal and no distress on completion of run.

30.9.35.—Animal killed and graft found in position. Arteries injected with cinnabar paste. (*Fig.* 453.)

Dog 2 (greyhound, young).—

21.3.35.—Operation: ligature of descending branch of left coronary artery.

28.5.35.—Operation: pedicled omental graft.

12.9.35.—Exercise tolerance tested on track. Running capacity normal, run finished in very good order.

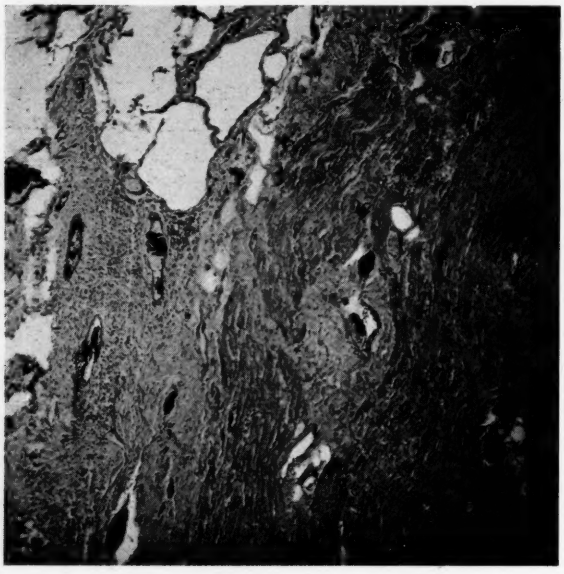

FIG. 454.—*Cat* 3. Injection of vessels in omentum and superficial layer of myocardium. (× 50.)

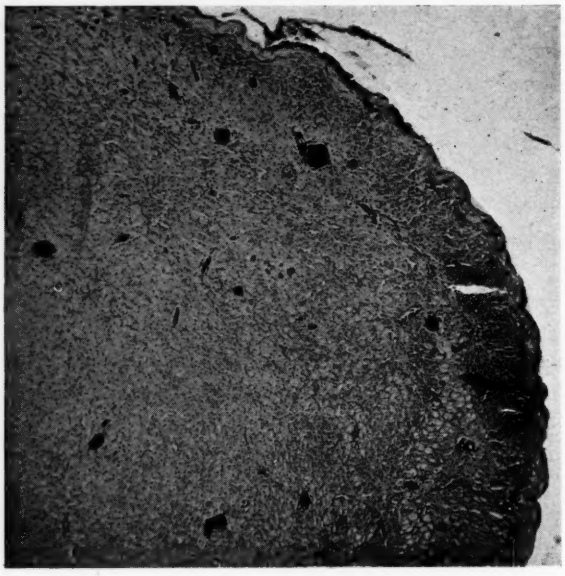

FIG. 455.—*Cat* 4. Injection of vessels beneath endocardium. (× 50.)

Cat 3.—

27.6.35.—Operation: pedicled omental graft.

24.9.35.—Killed and specimen injected (*Fig.* 454).

Cat 4.—

29.6.35.—Operation: ligature of descending branch of left coronary artery and pedicled omental graft.

25.9.35.—Killed and specimen injected (*Fig.* 455).

FIG. 456.—*Cat* 5. Injection mass lying in lymphatic spaces in myocardium. (× 50.)

Cat 5.—

16.2.34.—Operation: ligature of descending branch of left coronary artery.

16.7.35.—Operation: pedicled omental graft.

25.9.35.—Killed and specimen injected (*Fig.* 456).

A method has been described by which a collateral circulation to the heart may be established in the experimental animal. On injecting Indian ink suspension into the heart through the graft, particles of ink could be seen emerging from the cut aorta. In some of the sections vessels filled with ink may be seen lying immediately beneath the endocardium, and particles of the suspension are seen lying free in the chambers of the heart.

We have no data as to the length of time required for the new vascular connection to form, but in one animal killed three weeks after application of

the graft a very complete injection of the coronary arterial tree could be carried out.

At the beginning of this work some two years ago, it was intended to establish the functional importance of the pedicled omental graft in experimental coronary occlusion. For various reasons this intention has been abandoned. Observations on experimental coronary occlusion showed that many animals tolerated the procedure well, and the factors governing the occasional incidence of acute œdema of the lungs, pulmonary embolism, and acute heart failure remained unknown, so that a control group of animals could not be observed. At the same time the tests performed by the two dogs at least demonstrate that recovery from the operation is compatible with severe physical exertion.

I wish to thank the Council of the Royal College of Surgeons of England for providing the facilities for this work and the Medical Research Council for a grant in aid.

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SHORT NOTES OF RARE OR OBSCURE CASES

SARCOMA OF ACETABULUM MISTAKEN FOR TUBERCULOUS HIP

By C. H. FAGGE
CONSULTING SURGEON, GUY'S HOSPITAL

Leonard M., age 34, was first seen by Dr. A. H. Warde, of Purley, in April, 1934, when he complained of pain in the groin and the inside of the right thigh; the pain extended to the knee-joint. He walked with a limp, and examination of the hip-joint showed that the movement of abduction was limited; flexion and

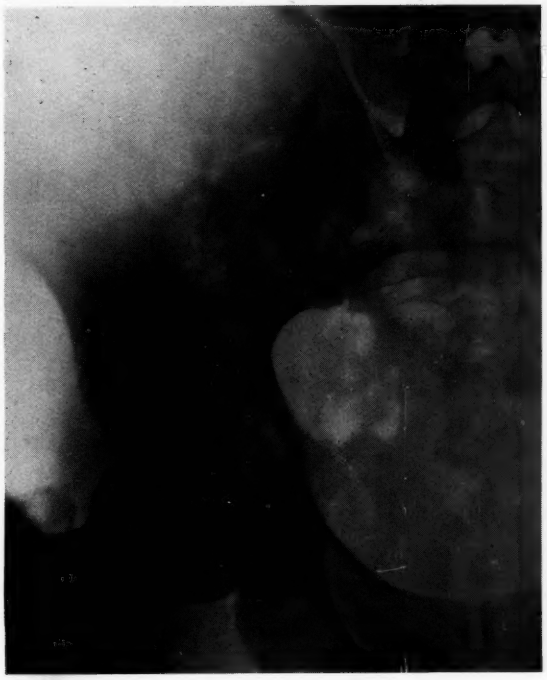


Fig. 457.—Right hip-joint when the patient was seen in June, 1934.

extension were normal. He was treated with rest in bed, but this had a strangely slight effect upon the pain.

When I first saw him in June, 1934, at the Purley War Memorial Hospital, I noted that the physical signs were strangely disproportionate to the severe pain

which he experienced. He was sent for X-ray report on the right hip-joint with a suggested diagnosis that the condition of the joint was tuberculous, but with the reservation that malignancy must be excluded. I think this alternative was suggested by the severity of the pain and by the fact that this pain was still marked, even though the patient had been resting for a month. The evidence in favour of tuberculosis of the hip was very strong, for the patient had had sanatorium treatment in childhood for tuberculosis of the lungs and mediastinal glands. He had had five children, all of whom had died in infancy from tuberculosis, according to the patient's statement.

Dr. Colyer made the following X-ray report on the right hip: "Large irregular area of rarefaction in bone above and on inner side of acetabulum, with thinning

FIG. 458.—Right hip-joint after death.

of bone socket about its centre. The bone surrounding the rarefaction is slightly sclerosed, suggesting an infective condition: tuberculosis is the most probable; does not suggest growth to me." (*Fig.* 457.)

With the assistance of Dr. A. H. Campbell, Tuberculosis Officer to the Surrey County Council, the patient was admitted to the Royal Sea Bathing Hospital, Margate, on June 29, and from that date Dr. B. W. Armstrong, Medical Superintendent, has been good enough to report on his case as follows:—

"On admission the right hip was in a position of slight flexion and adduction. There was a small swelling, (?) abscess, on the anterior surface of the upper thigh; enlarged inguinal glands; local heat; pain on attempted movement but not particularly severe. There were some coarse breath-sounds and areas of increased

vocal resonance in the upper left chest. Clinical evidence of some scattered fibrosis. His temperature was normal or subnormal from the date of his admission to September, when, four days after the patient was fixed in an abduction frame, his temperature rose to 101° and remained about that level for over two months, when it gradually fell and remained subnormal until his death. There was nothing to arouse suspicion of any other diagnosis than tuberculosis, particularly as our radiologist reported that there was chronic pulmonary tuberculosis of the right apex and tuberculosis of the upper part of the acetabulum and ilium above it, with a fair-sized abscess lying lateral to the joint. He was put up in extension, but the infiltration and induration involving the upper part of the thigh increased and he was suffering very severe pain. This was not relieved by an abduction frame, and in the hope of evacuating a deep-seated abscess an aspirating needle

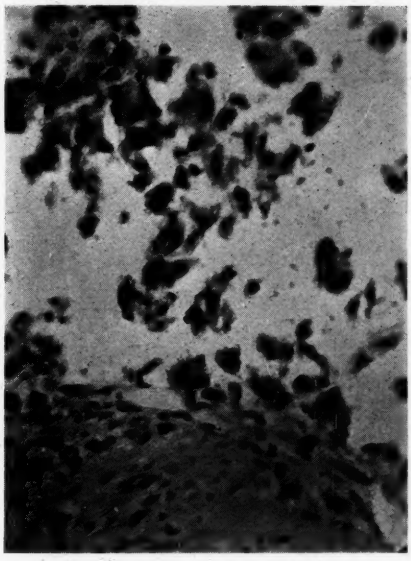


FIG. 459.—Post-mortem section of growth.

was put in the swelling on two occasions without finding pus. On the second occasion a small piece of solid material was sucked into the syringe, which microscopically was reported to be a mixed-celled sarcoma.

" He continued to go downhill and required enormous doses of morphia to relieve his pain. The X-ray taken post mortem (*Fig.* 458) shows an extensive destruction of the ilium, which was verified at the autopsy, and there were no secondary deposits discovered in the chest or abdomen."

The Clinical Research Association reported on the small fragment removed by an aspirating needle and on a post-mortem section of the growth that it was a cellular atypical giant-celled sarcoma. Mr. Davies-Colley has been good enough to examine the section, and reports that it shows " the usual polymorphic type of osteogenic sarcoma of bone and suggests a very high degree of malignancy " (*Fig.* 459).

METAPHYSIAL ACLASIS WITH TUBERCULOUS INVOLVEMENT OF A JOINT

By A. CAMERON ARMSTRONG, Liverpool

The following case is presented as having two unusual features of interest—namely, the occurrence of tuberculosis in a joint the site of the exostoses typical of metaphysial aclasis, and the extensive involvement of its synovial membrane with tuberculosis without appreciable loss of movement or deformity of the joint, without radiographic bone changes, without pain, and, as far as could be seen at biopsy, without involvement of articular cartilage or bone with the disease. While Hale,[1] in a consideration of 50 cases of metaphysial aclasis, describes tuberculosis as the commonest associated disease, he makes no reference to its special localization

Figs. 460, 461.—Radiographs of left knee showing presence of bony outgrowths (August, 1934).

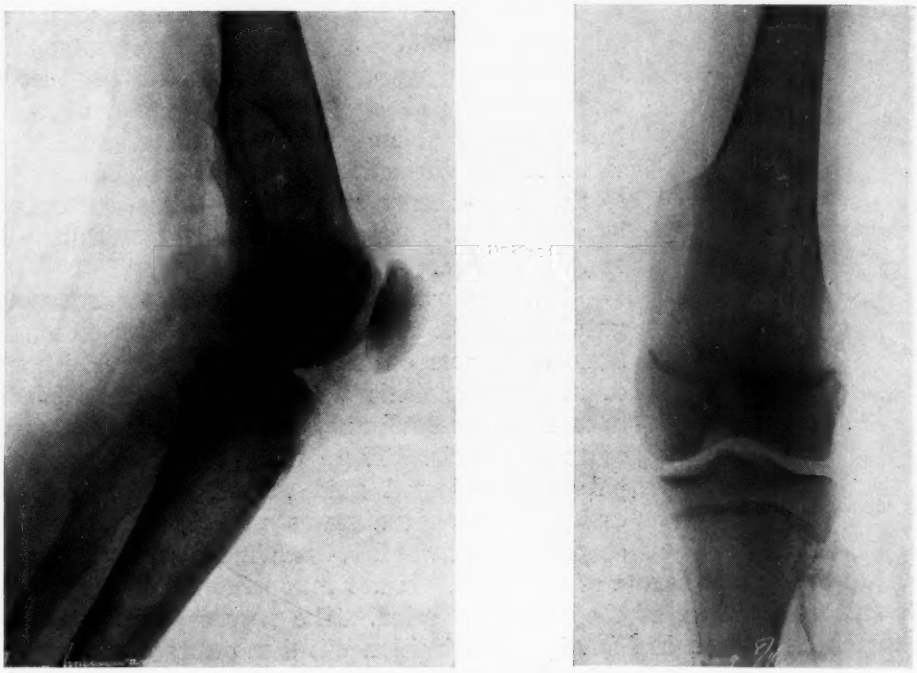
in joints. The hereditary factor which forms such a prominent feature of his cases, and of others, was not obvious in the one here described.

M. M., aged 12, schoolgirl, daughter of working people, first attended for examination on Aug. 23, 1934, stating that the previous day she had noticed a swelling of the left knee, and was experiencing difficulty in getting downstairs. There was no trauma, and no history of infectious disease or other illness. Her general health was said to be good and she was of normal height and weight. There was no history of cough or chest trouble present in any member of the family.

Examination showed a painless effusion into the left knee. The movements of the joint were full, apart from the last few degrees of flexion limited by the

effusion. There was no wasting of the thigh. X-rays of the left knee showed the presence of bony outgrowths characteristic of metaphysial aclasis, but no evidence of tuberculous involvement of the joint (*Figs.* 460, 461). X-rays of the other knee showed a similar condition. Further radiograms of the ankles, wrists, elbows, and hips, showed no abnormality. The urine was normal. The family history contra-indicated congenital syphilis. She was sent to the massage department for quadriceps drill, and the effusion into the knee gradually became less, while the thigh did not waste.

On Oct. 16 there was no effusion, but a definite thickening of the synovial membrane below the patella. This had increased markedly by January, 1935, when she was admitted to hospital. At this time she was anæmic, and not looking



Figs. 462, 463.—Lateral and antero-posterior radiographs of the knee taken in January, 1935.

well. Wassermann and Kahn tests were negative. Further X-rays of the knee at this time (*Figs.* 462, 463) showed no evidence of tuberculosis, as did the X-ray of the chest, but owing to the absence of improvement an arthrotomy was performed, and the synovial membrane of the joint was found to present the characteristic appearance of tuberculous infection. The articular cartilage and the portions of bones that were visible were normal in appearance. Microscopic sections of the synovial membrane confirmed the presence of tuberculosis. A Thomas splint was applied to the limb, and the child made very rapid improvement in her general health. No other member of her family, as far as is known, has a condition of metaphysial aclasis.

REFERENCE

[1] HALE, *Ann. of Surg.*, 1930, xcii, 92.

A RETROPERITONEAL ENTEROGENOUS CYST

By LOUIS J. HORN

LATE RESIDENT SURGICAL OFFICER,
GLOUCESTERSHIRE ROYAL INFIRMARY, GLOUCESTER

ENTEROGENOUS cysts, though not extremely rare, are sufficiently so to be worth recording. The following case showed some unusual features as regards form and position of the cyst.

A male child, C. J. B., aged 4, was admitted to the Royal Infirmary, Gloucester, on June 21, 1934, with a history of abdominal pain coming on in spasms, during two of which the child had vomited. The symptoms had been present for twenty-four hours and had become worse after the giving of a dose of castor oil, which had produced several actions of the bowel. There was nothing distinctive about the vomitus or the stools. No history of previous similar attacks was obtainable. The temperature was 98·4° and the pulse 124 per minute.

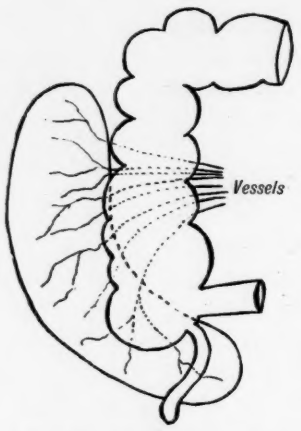


Fig. 464.—Condition found at operation.

On examination there was much tenderness and guarding in the region of the right iliac fossa, and some tenderness on rectal examination. A diagnosis of acute appendicitis was made, and the abdomen opened by the McBurney muscle-split incision under ether anæsthesia. There was a moderate amount of pale straw-coloured fluid present in the peritoneal cavity. The appendix was normal, but a sausage-shaped tumour was felt, extending from the mid-pelvis almost to the hepatic flexure of the colon. It was retroperitoneal and lay parallel with and to the right of the ascending colon. The cæcum partly overlapped the tumour, as shown in *Fig.* 464. The incision was then enlarged to the extent of 1 in. in an upward direction and ½ in. in a downward direction. The pelvic end of the cyst was lightly adherent by fresh lymph to the surrounding structures. The peritoneum was incised between cyst and colon, and easily stripped from the former, which was then freed. A flat leash of vessels of supply entering its upper part from beneath the ascending colon was clamped and ligated, and the cyst removed. Beyond some areolar tissue there was no connection of any sort between the cyst and the colon. The child's condition was good at the completion of the operation, and he made an uneventful recovery.

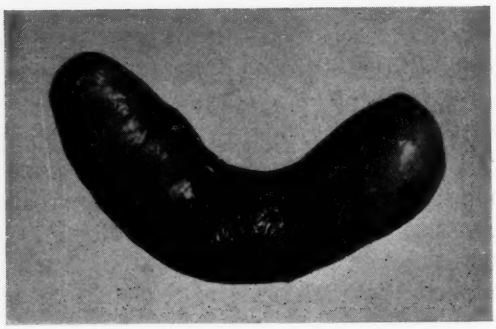


Fig. 465.—Appearance of the cyst after removal.

Examination of the cyst showed it to be 7 in. in length, and 2 in. in diameter. It was sausage-shaped (*Fig.* 465) and semi-translucent in appearance. The wall closely resembled that of small intestine, and had well-marked outer longitudinal and inner circular layers of muscle. There was no peristaltic movement visible as has been described in some such cysts after their removal. The cyst was carefully opened at one end, and found to contain clear fluid. A microscopic

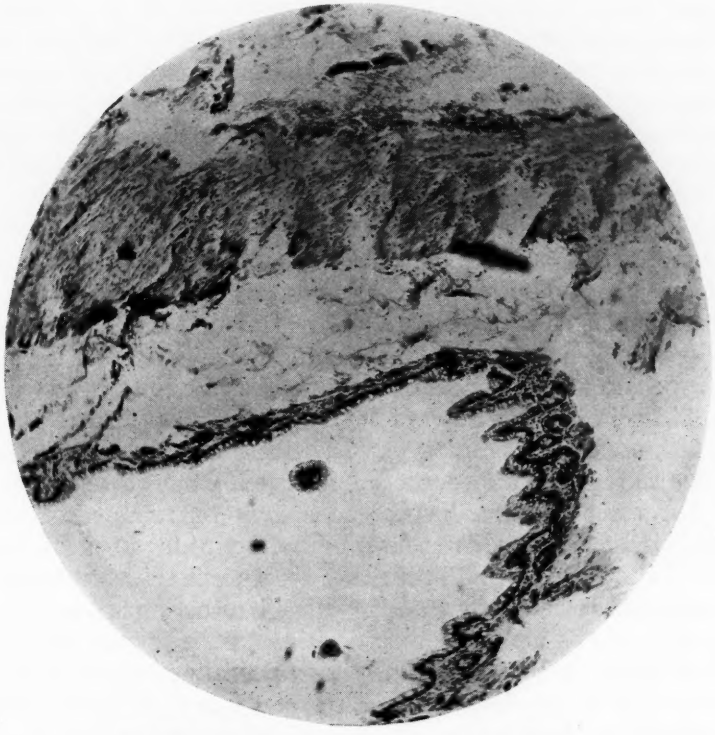


Fig. 466.—Microscopic section of the wall of the cyst.

section of the wall and an examination of the fluid were made by Dr. E. N. Davey, pathologist to the institution. The section shows two well-marked muscular layers and a poorly developed or atrophic mucosa (*Fig.* 466). The mucosa shows simple villi covered by columnar epithelium containing numerous goblet cells. The fluid was of low specific gravity, and contained mineral salts and protein, but no intestinal ferments.

COMMENT

Congenital cysts and diverticula are known to occur in practically all parts of the alimentary tract. The greater number of those which have been described reproduce more or less completely the structure of the gut, thus indicating their origin. The type of epithelium may be no guide as to which portion of the gut they came from, since several factors tend to modify this—viz., changes brought about by tension and inflammation, and heteromorphic changes due to the abnormal environment of such epithelium—cf. the common finding of gastric mucosa or

pancreatic tissue in Meckel's diverticula. There are two modes of origin of these cysts which reproduce bowel structure :—

1. Those which arise in connection with some unobliterated portion of the vitello-intestinal tract. There may be a fibrous persistence of this tract, with a cyst along some part of its course. The tract may not be represented along its whole length, but a cyst occur on the antimesenteric border of the gut in the position of the more usual Meckel's diverticulum, or underneath the peritoneum deep to the umbilical scar.

2. Those which arise in connection with diverticula of the developing gut.

The work of Lewis and Thyng[1] in particular throws light on this mode of origin. They examined the developing gut of many rabbit and pig embryos of about 5 to 20 mm. in length. Many of these showed vacuolation and diverticula in the endoderm, especially in the region of the jejunum and ileum. Some of these diverticula were seen to pierce the developing muscularis, and in one pig embryo there was an epithelium-lined cyst outside the muscularis, and a solid cylindrical outgrowth of the gut epithelium adjacent; connection had been lost between the two. Their researches suggest that these cysts have their origin in this way. It will readily be understood that such a piece of sequestered endoderm may come to occupy almost any position in the abdomen; either between the layers of the mesentery, or behind the peritoneum. Also, since it has to traverse the muscularis, it may be held up in between any two layers, and develop in that position. This latter is, indeed, a common position in which these cysts are found.

The ileocæcal region is a favourite site for enterogenous cysts; they are usually intramural, but some are free in the mesentery or retroperitoneal. Cases of so-called reduplication of the bowel have been described. In these is seen a tube or a cyst parallel to the bowel. There may be a communication of the lumina or merely a firm union of the walls; one or other is usually present, thus indicating that these reduplications are really diverticula that have grown along parallel with the bowel from which they sprung. They are essentially the same as the intramural ones, and those which have become more remote from their parent bowel. These forms are mentioned because the cyst under discussion was elongated in shape, and lay more or less parallel to the colon. It does not, however, I think, enter this category, since it was free of the neighbouring colon, and, both in naked-eye and microscopic structure resembled small intestine. It is, apparently, one that has become farther separated from its source of origin than is usual.

The mild inflammatory state of its lower end probably caused the symptoms and signs, since it doubtless caused an increase of fluid and therefore of tension in the cyst. There was no evidence of obstruction to the bowel by pressure, which is the usual mechanism by which these cysts cause symptoms; more especially in the case of the intramural ones. Many of them do not cause symptoms at all, but have been found accidentally at autopsy.

I am indebted to Mr. Arnold Alcock for permission to publish this case.

REFERENCE

[1] LEWIS and THYNG, *Amer. Jour. Anat.*, 1907–8, vii, 505.

A CASE OF INTRACRANIAL PNEUMATOCELE

By A. E. PAYNE and W. D. JEANS, Leicester

References in the literature to the presence of air in the cranial cavity as a complication of a fracture of the skull are fairly numerous, but the similar entry of air as the result of the impairment of the integrity of the skull by an osteoma of the orbito-ethmoidal plate is much more rarely recorded, only two previous references having been found in the literature. The first of these is by Harvey Cushing[1] and is described by him as "a large intracranial pneumatocele of unexplained origin exposed and emptied at operation; recurrence of pneumatocele; second operation revealing a minute pneumatic sinus alongside an orbito-ethmoidal osteoma; closure by fascial stamp; recovery". The second is recorded by F. Gordon Bell,[2] and the condition was first revealed by a highly characteristic X-ray picture, upon which the radiologist, Dr. Barclay, reported: "There is an osteoma arising from the orbital plate of the right frontal bone close to the mid-line and apparently encroaching on the frontal sinus. There is an extensive right pneumocephalus with displacement of the falx and compression of the brain substance. The air probably lies in the subdural space". The air was found to be in the brain substance at operation, when, as in Cushing's case, the osteoma was successfully removed.

In addition to these two cases Armitage[3] published a somewhat similar case of Cushing's in which there was an osteoma of the frontal sinus with a mucocele communicating with the ventricular system.

Dandy[4] made an exhaustive study of the literature of air inside the skull cavity, which he called pneumocephalus (intracranial pneumatocele or aerocele). All the cases he was able to collect were due either to trauma or to infection (except one case reported by Santoro as being due to a tumour eroding the floor of the skull, but no notes of the case were available) and he says that the condition was due to fracture of the skull affecting the frontal sinus or the ethmoidal or mastoid air-cells, or else due to infection with a gas-producing organism.

He says: "The air may be situated in: (1) the subdural space, (2) the subarachnoid space, (3) the brain, (4) the ventricles, and of these the most common is the intracerebral variety. The entrance of air must occur relatively late, after the dura has become adherent to the traumatized brain, and possibly rupture of the cerebral cortex at the time of injury is also necessary. The anatomy of the frontal region is favourable to the production of intracerebral pneumatocele, first because the dura is so closely applied to the frontal bone that a fracture readily tears the dura, and second because the tip of the frontal lobe is in such close apposition to the dura that the cerebral cortex as well as the meninges is easily ruptured. Even so, it is doubtful if an intracerebral pneumatocele could develop without an added factor, i.e., the increased pressure of air within the cranial sinuses such as is produced by sneezing, coughing, straining or possibly even swallowing."

Our case was that of a gasworks labourer aged 58 years. It was impossible to obtain any history from the man himself, and his relatives could not give a

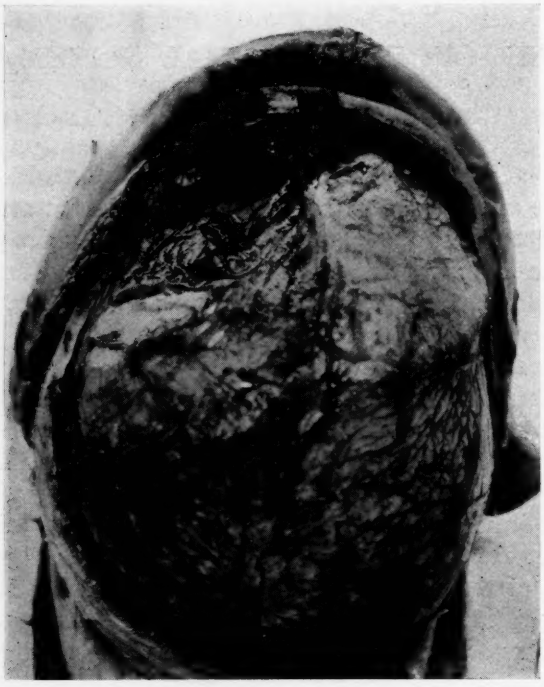


FIG. 467.—The brain taken from above, showing the extent to which the frontal lobe could be collapsed by withdrawing air, and the osteoma below the frontal pole.

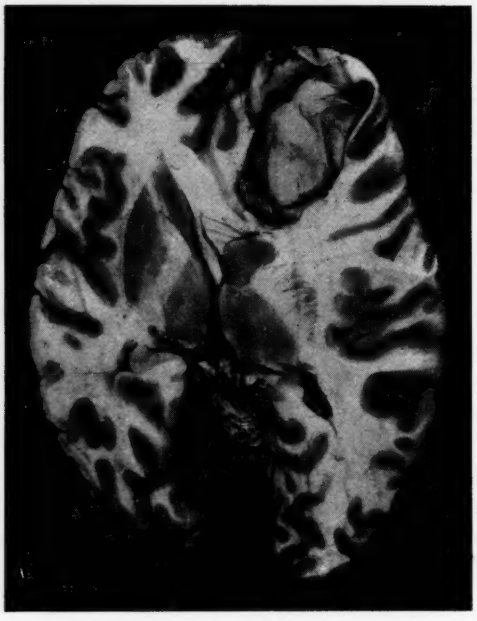
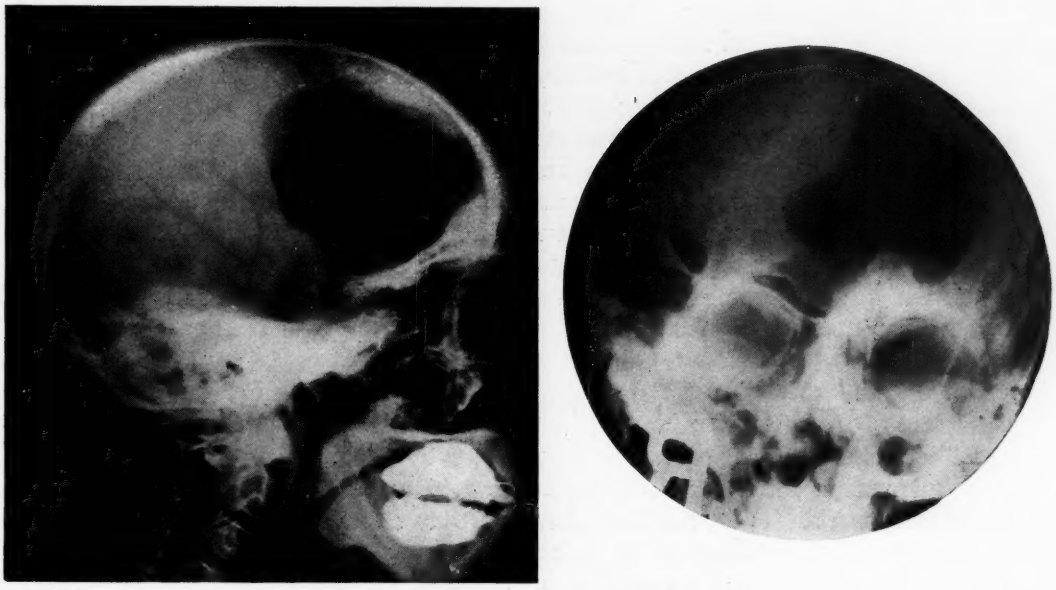


FIG. 468.—A section of the brain showing the extent of the cavity and its relation to the caudate nucleus and the internal capsule.

satisfactory detailed account. He had complained of headache (apparently his only complaint) for three years, but was able to follow his occupation until he was knocked down by a bicycle six weeks before admission. He was apparently uninjured by this mishap, but shortly afterwards began to lose his memory for recent events.

On Admission.—The man was unable to give any account of himself, and was not very co-operative during examination; he could only answer 'yes' or 'no', and then not always in the right context. There was weakness of the whole of the right side of the body, with increase of the deep reflexes on the right side, but with normal plantar responses. It was impossible to test sensation in any way. There were no signs of any increase in intracranial pressure, the fundi being normal and the cerebrospinal fluid pressure being only 70 mm. of water. The cerebrospinal fluid itself showed some change from the normal, cells being increased to 7 per c.c.,



Figs. 469, 470.—X-ray appearances.

protein content to 70 mgrm., and the gold curve altered to 1223221000. The report on the skull X-ray said: "The left frontal sinus is occupied by a dense bony mass, and there is a large collection of air in the left frontal region, apparently communicating with the frontal sinus."

Operation.—Two days after admission he became gradually unconscious, with a complete right-sided paralysis and a right extensor plantar response. As the coma gradually deepened during the next two days, Mr. Barrett turned down a left frontal flap. The dura was found to be very adherent over the frontal lobe; the aerocele was aspirated, and the osteoma could then be seen below the collapsed frontal lobe. Owing to the patient's poor condition nothing more could be done, but it was hoped that relief of pressure would procure a return to consciousness. Unfortunately this did not happen and the patient died the following day without any return to consciousness.

POST-MORTEM EXAMINATION.—Autopsy showed an osteoma completely destroying the roof of the left frontal sinus. Immediately above this there was an opening in the dura and in the base of the frontal lobe measuring 1 cm. in diameter. This opening led directly into a cavity measuring 6½ cm. antero-posteriorly, 3 cm. from side to side, and 4 cm. in depth, and stretching back almost as far as the caudate nucleus. (*Figs.* 467, 468.)

Microscopical sections taken from different places along the cavity all showed normal brain tissue ceasing abruptly at the cavity.

The X-ray appearances (*Figs.* 469, 470) are so startling and unexpected that any failure to accept the obvious intrepretation as the right one can be more readily understood than excused, this obvious interpretation being that, not only are the appearances due to a collection of air, but that the air is intracerebral, and neither intraventricular nor subdural. As against the former there is no sign of the normal outlines of any of the ventricles, and as against the latter the position and shape of the collection, especially as viewed in coronal section, are so much those of the cerebral hemisphere itself that an equal wall of brain tissue around it seems certain. That the air is under pressure is evident, and it is the interplay of this pressure and the resistance of the surrounding structures which result in the bulge across the mid-line in the lower part where the restraining influence of the falx is no longer present. At only one part of its containing wall does the collection of air become superficial, and that is the point where the frontal lobe abuts on the frontal sinus. The sinus itself is seen to be completely occupied by a dense osteoma which is tending also to extend into the ethmoid region. The close association of the pathological air-cell with the aerocele could hardly be other than one of cause and effect.

DISCUSSION

The similarity of our case to those described by Cushing, and to that described by Bell, is very striking. It would appear that air may enter the brain substance in one of three ways: (1) By the formation and rupture of a cerebral abscess; (2) By the formation and discharge of a cerebral mucocele; (3) By the admission of air through the destruction of the roof of one of the sinuses either by a fracture or by a tumour. In our case the roof of the frontal sinus was completely destroyed and the dura and cerebral cortex were very adherent, both to each other and to the bone. The walls of the cavity in the brain were composed of normal tissue, so that it seems probable that the aerocele was caused by a sudden increase in pressure following nose-blowing or some such other simple procedure, and by a splitting of the brain tissues along the natural layers of cleavage.

The symptomatology and clinical findings in this case were very suggestive of a frontal tumour, and the true diagnosis was only made possible by X rays, which it is very important to employ in the routine investigation of all intracranial conditions.

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A CASE OF RETROGRADE INTUSSUSCEPTION OCCURRING DURING LIFE

By EMLYN E. LEWIS
RESIDENT SURGICAL OFFICER, QUEEN'S HOSPITAL, BIRMINGHAM

A WOMAN, aged 47, was admitted to this hospital on Aug. 30, 1935, with a history of colicky abdominal pains of three days' duration. For twenty-four hours prior to admission she had vomited on a number of occasions. Her bowels had not been opened for three days, neither had she passed flatus. She had noticed increased difficulty with her bowels over the last three months. Her pulse-rate was 120, her temperature subnormal, her abdomen very much distended, especially over the left side, and tympanitic all over. A diagnosis of intestinal obstruction due to volvulus was made.

OPERATION.—On opening the abdomen under spinal anæsthesia, the transverse colon was found to be greatly distended and felt very thickened. This distension and thickening continued back to the cæcum, where the thickening ended in a hardish mass situated at this point. Only a few inches of collapsed pelvic colon could be felt, and on tracing this upwards it was found to enter the gut proximal to it and a few inches distal to the splenic flexure. One realized immediately that this was a retrograde intussusception and that the mass at the cæcum was the apex. The intussusception was reduced by the usual manipulations, some difficulty being experienced in reducing the apex. After reduction, the apex of the intussusception was found to be that part of the pelvic colon opposite the longest portion of the pelvic mesocolon. The apex of the intussusception was gangrenous over about 8 in. The transverse and pelvic colons were markedly elongated, the lumen of the latter being unusually large with its walls very thickened. The pelvic mesocolon, too, was elongated, being about 20 in. long opposite that part of the pelvic colon which formed the apex of the intussusception. The gangrenous loop was treated on the lines of Paul's operation, being delivered on to the abdominal wall via a left iliac incision. The operation was completed by tying a Paul's tube into the gangrenous portion.

SUBSEQUENT HISTORY.—Twenty-four hours after operation the patient's general condition was highly satisfactory and the colostomy was working very well. This remained so until the fourth day, when she developed signs of a right-sided pneumonia, from which she succumbed on the sixth day following operation. Unfortunately a post-mortem examination was refused.

Comment.—The case has been recorded because of the rare condition found at operation. Retrograde intussusceptions are said to occur only in the throes of death, and then as a rule only in the small intestine. Occasionally in life intussusception may take place at a gastro-enterostomy opening. Lockhart-Mummery states, "Retrograde intussusceptions do occur but only during death or as a result of asphyxia; they are not met with in practice." The condition in the case recorded above must have been present for three days at least, and but for the onset of pneumonia the patient would have made a good surgical recovery. At no time was there any evidence of peritonitis.

LEFT DIAPHRAGMATIC HERNIA: PERFORATION OF GASTRIC ULCER INTO LEFT PLEURAL CAVITY

By A. H. SANGSTER, Birmingham

The following case is presented more for its pathological interest than for its clinical importance.

James H., a widower, aged 64 years, was admitted to hospital in emergency as a case of perforated gastric ulcer. He gave a history of the sudden onset of acute

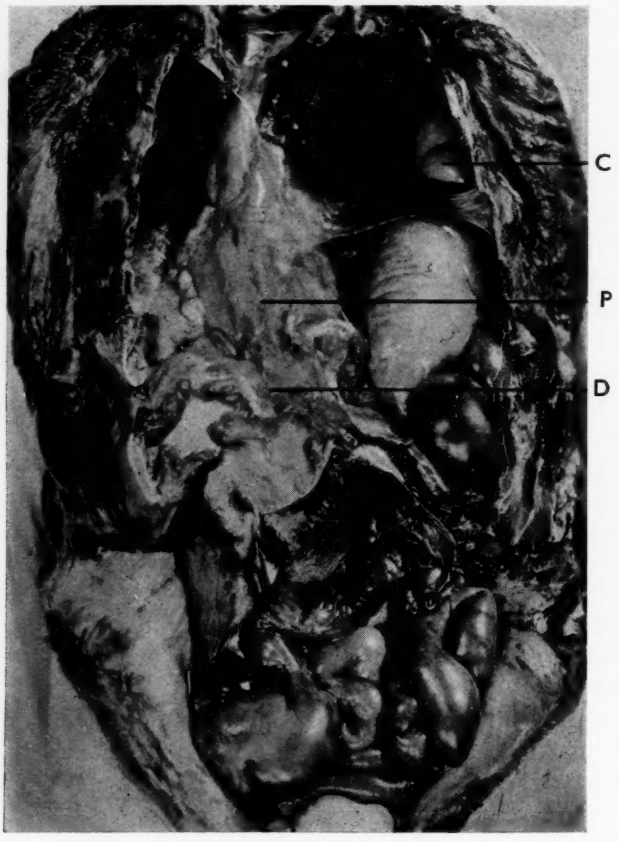

Fig. 471.—Condition found post mortem. D, Diaphragm; C, Apex of loop of colon; P, Pericardium.

abdominal pain four hours previously, starting in the left side of the epigastrium and rapidly becoming more widespread. He denied any previous ill health and gave his age wrongly, but was in extreme pain and was confused, having been given morphia by his doctor before admission. Later inquiries, however, showed that he had been in hospital twice before and the diagnosis of diaphragmatic hernia made.

ON EXAMINATION.—The patient appeared a wasted old man in extreme pain with a temperature of 99°; the respirations were very shallow and slow, four per minute. No abnormal sounds were heard in the chest, the heart-sounds were weak and in the normal position, the pulse was imperceptible. The abdomen appeared full with general board-like rigidity.

OPERATION.—His condition failed to improve with $\frac{1}{4}$ gr. of morphia and a heat cradle, so laparotomy was performed half an hour after admission, under general anæsthesia. A left paramedian incision was made and some dirty free fluid evacuated; there was great difficulty in locating the stomach, which was found very high up and overlaid by the transverse colon. The ulcer was found, the perforation being apparently on the posterior surface. The patient's condition became extremely grave and prevented further procedure, so the abdomen was rapidly closed by through-and-through sutures. He died shortly after.

POST-MORTEM EXAMINATION.—Autopsy was performed by Dr. Robinson, who found: General peritonitis; left diaphragmatic hernia; perforation of gastric ulcer into the left pleural cavity; purulent pericarditis; and arteriosclerosis.

The body appeared that of a wasted, grey-haired edentulous man. Fibrinous effusion, 2 pints, containing stomach contents, in left pleural cavity; oval deficiency 8 × 6 cm. in tendinous portion of left dome of diaphragm, edges smooth and fibrous, no hernial sac. Loop of transverse colon 30 cm. long, stomach with greater curve above and posterior surface forwards, and 25 cm. of dilated jejunum lying in left pleural cavity. Collapse of left lung; loop of colon crossed by fibrous band, 2 cm. broad, running from lower border of left upper lobe to parietal pleura. Fibrinous adhesions between lesser curve of stomach and left border of pericardium; purulent pericarditis with effusion, 3 oz.; perforation, 1 cm. diameter, on anterior aspect of lesser curve of stomach in floor of an ulcer, 3 cm. diameter, lying astride lesser curve 4 cm. from pylorus. Fibrinous peritonitis with turbid effusion, 1 pint; dilatation of jejunum. Phrenic nerve identified running downwards to inner border of hernial orifice, then backwards to œsophageal opening. Congestion and œdema of right lung. Brown atrophy of myocardium; area of fibrosis 2 × 2 cm. in apex of left ventricle; severe atheroma, especially of coronary arteries. Arteriosclerotic kidneys. Brown atrophy of liver. Remainder of post-mortem showed nothing of importance. (*Fig.* 471.)

REVIEWS AND NOTICES OF BOOKS

The Stomach and Duodenum. By GEORGE B. EUSTERMAN, M.D., F.A.C.P., Head of Section in Division of Medicine, the Mayo Clinic; DONALD C. BALFOUR, M.D. (Tor.), LL.D., F.A.C.S., F.R.A.C.S., Head of Section in Division of Surgery, the Mayo Clinic; and Members of the Staff of the Mayo Clinic and Mayo Foundation. Large 8vo. Pp. 958 + xv, with 436 illustrations. 1935. Philadelphia and London: W. B. Saunders Co. 45s. net.

THOSE who have had the good fortune to visit the Mayo Clinic have realized the vastness of the opportunities there offered for clinical and pathological research upon practically every branch of medical science, and the marvellous organization of team work by which these unique opportunities are utilized to the utmost possible advantage. We heartily welcome, therefore, this monumental work upon the medical and surgical aspects of the stomach and duodenum written by the Heads of the Sections of Medicine and Surgery respectively—two such well-known authors as Dr. Eusterman and Dr. Donald C. Balfour—with the collaboration of thirteen other members of the Mayo Clinic Staff. While founded mainly upon the actual work of the Clinic itself, yet it is evident that an exhaustive search of the literature upon the subject has been carried out and the results incorporated, while everywhere full recognition is given to English and foreign investigators. So completely has the ground been covered, and so thoroughly up to date is this volume, that we have failed to find any fact of importance in connection with the stomach and duodenum to which adequate reference has not been made: it is indeed obvious that for a long time this will be the standard work of reference on diseases of these viscera.

A very readable account of the history of these diseases by Dr. Dwight L. Wilbur is followed by a chapter upon the applied physiology of the stomach and duodenum by Dr. Walter C. Alvarez, wherein all the bearings of experimental physiological research upon their functions are considered in a masterly and thoroughly practical manner. Stress is laid upon the act of swallowing, especially of solid food, as being Nature's one big stimulus to peristalsis, and it is pointed out that some of the paretic distension of the bowels that occurs after many operations is due to the enforced fasting so often insisted upon. In this connection a sphere of usefulness is found for the American habit of chewing gum. Dr. F. C. Mann, whose fame as an experimental investigator is world-wide, writes fully upon the experimentally produced chronic gastric and duodenal ulcer, and shows that practically every aspect of the formation and healing of these ulcers as met with clinically can be reproduced and studied in the experimental animal. This has entailed an enormous amount of experimental work, the results of which are concisely stated, while some excellent reproductions are given of experimentally induced ulcers and their healing.

The section on the surgical pathology of the stomach and duodenum by Dr. W. C. McCarthy details the findings of the careful investigation during the years 1899 to 1933 inclusive of no less than 4129 gastric and 1589 duodenal specimens, all of which were removed during the course of a surgical operation. The subject of 'dyspepsia' is dealt with most exhaustively under the four headings of 'organic', 'reflex' 'systemic', and 'functional', and the description of the methods of clinical investigation of the patient indicates the thoroughness and care with which these examinations are carried out routinely. Gastroscopy by the Wolf-Schindler flexible gastroscope has not yet been generally adopted at the Clinic. At the top of page 142, in referring to Morley's work upon the mechanism of deep tenderness in gastric ulcer, it is stated—"Morley concluded that the tender point was situated in the anterior part of the parietal peritoneum, that is, that it was a true visceral tenderness": it is obvious that these two statements are incompatible, the first alone correctly stating Morley's opinion. The chapter on roentgenologic diagnosis is excellent and deals not only with the

appearances of the stomach and duodenum in the normal and in diseased states, but describes fully the appearances that are to be regarded as 'normal' after the successful performance of the various surgical operations upon these viscera, and also as 'abnormal' when post-operative complications supervene—e.g., gastrojejunal ulcer.

The subject of duodenal ulcer is discussed thoroughly in all its aspects, and the indications for medical and surgical treatment respectively are most judiciously considered, while each step of the various operative procedures applicable is concisely described and splendidly illustrated. An unusual feature is a chapter on 'duodenitis' as a precursor and as an accompaniment of duodenal ulcer, while in other chapters the rarer lesions of the duodenum—benign and malignant growths, tuberculosis, fistulæ, parasites, diverticula, paraduodenal herniæ, injuries, and foreign bodies—are brought fully up to the present state of our knowledge.

The surgical treatment of gastric ulcer and gastric carcinoma is magnificently described and profusely illustrated, and in no other work can such a complete and excellent account of that very distressing post-operative sequel—anastomotic ulcer—be found. The conclusions arrived at as to its surgical treatment are that if the original duodenal ulcer has healed without producing any obstruction, the operation of choice is disconnection of the anastomosis and nothing more, while, should the ulcer be still active or have produced obstruction, the best course is partial gastrectomy, preferably with union by the Billroth I method. It is a sad reflection upon surgical energy and judgement that it has been found necessary to write a section upon 'gastro-ileostomy' and its sequel in 'ileac ulcer'. Attention is drawn to the almost complete limitation of gastro-jejuno-colic fistula to the male sex, there being but one female out of 74 cases observed at the Mayo Clinic. Post-operative complications are so fully described that it is difficult to conceive of any emergency arising after operation upon the stomach or duodenum that is not fully dealt with in this volume.

Typical diet-sheets for the pre- and post-operative treatment of gastric and duodenal ulcer and gastric carcinoma are given in the appendix.

This volume is thoroughly well calculated to maintain the world-wide and exceedingly high prestige already attained by the Mayo Clinic.

Diseases of the Rectum and Colon and their Surgical Treatment. By J. P. LOCKHART-MUMMERY, M.A., M.B., B.C. Cantab., F.R.C.S., Senior Surgeon to St. Mark's Hospital. Second edition. Large 8vo. Pp. 616 + viii, with 247 illustrations. 1934. London: Baillière, Tindall & Cox. 35s. net.

WE welcome a second edition of this excellent work. A period of eleven years has elapsed since the publication of the first edition, during which time, as the author points out in the preface, considerable advances have been made in our knowledge of the surgery of the rectum and colon. As the result, a large part of the book has been entirely re-written and several new chapters have been added. In addition, ninety new illustrations have been included, chiefly photographic reproductions of the technique of actual operations.

The first part of the book contains chapters on diseases of the rectum and anus; the remainder is devoted to diseased conditions of the colon.

The opening chapter gives a succinct account of the anatomy of the rectum and colon. Then follows an account of the methods of examination of a patient suffering from rectal disease, stress being laid upon the necessity of carrying out an examination by means of the sigmoidoscope in every instance in order to determine whether diseased conditions exist beyond the reach of digital examination. The author's method of preparing a patient for operation and the details of the after-treatment which he adopts are set forth in the succeeding chapter. The subject of hæmorrhoids is fully discussed, the various methods of treatment receiving careful consideration. The technique of Whitehead's operation is described in detail, but the method is not recommended as the results usually obtained by the generality of surgeons are not good. In the following chapter the subject of prolapse of the rectum is dealt with at length and the technique of the author's method is strongly advocated as yielding excellent results. Abscess, fissure in ano, and the various inflammatory processes encountered in the rectum are elaborately discussed. Two chapters are devoted to the subject of fistula and its surgical treatment, much useful information being incorporated therein. The classification of fistula is based upon causation, no mention being made of the relationship of the original

abscess cavity to the muscular and connective-tissue planes in determining the various types that are met with. The author lays great stress upon the effect of division of the external sphincter muscle in the production of an incontinent anus following operations for fistula. In those instances in which the main track passes beneath the muscle he is emphatic in his opinion that the muscle should not be divided at the primary operation, but should be dealt with at a later stage when the greater part of the original wound has healed. As the result of having been extremely careful to avoid injury to the external sphincter he says: "I can confidently assert that, although I have operated for fistula a great many times, I have never produced an incontinent anal opening, and there are no patients of mine walking about this earth cursing their misfortune and my incompetence on this account."

We are constrained to think that such a splendid record is not solely due to the avoidance of division of the external sphincter, because we are not aware that incontinence results from the operation usually adopted for the cure of a fissure which entails complete division of the muscle.

Chapters devoted to the consideration of pruritus ani, rectal pain and neuralgia, simple tumours of the rectum and colon precede a very interesting discussion of precancerous conditions. The author believes that cancer only occurs in those who have inherited a genetic susceptibility to mutation of the cells in certain tissues, and that the susceptibility is to hyperplasia of the cells. As a result of hyperplasia adenomata are formed which not uncommonly eventuate in cancer. The subject is a fascinating one and well worthy of further investigation. Cancer of the rectum is elaborately discussed from the etiological, symptomatological, pathological, and surgical points of view. When describing the methods of extension of rectal growths, the impression is conveyed that the determination of the three paths of spread, namely, (1) by infiltration of surrounding parts through continuity of tissue, both on the surface and deep to it, (2) by the lymphatics, (3) by the veins, was due to an investigation carried out in the Cancer Research Department of St. Mark's Hospital by Dr. Cuthbert Dukes and reported by him in an article entitled "The Spread of Cancer of the Rectum" which appeared in the BRITISH JOURNAL OF SURGERY in 1930. It seems to be within our recollection, however, that these modes of spread were originally described in a paper read in the Section of Surgery at the Annual Meeting of the British Medical Association held at Cambridge on July 1, 1920, and which was published in the *British Medical Journal* of Nov. 13 of that year.

The remainder of the book is concerned with abnormalities and diseases of the colon and their treatment.

We have pleasure in commending the book as representing the opinions of one who has spent many years in the practice of rectal surgery.

An Introduction to Surgery. By RUTHERFORD MORISON, M.D., F.R.C.S. (Edin.), F.R.C.S. (Eng.), M.A., D.C.L., LL.D., Emeritus Professor Surgery, Durham University; and CHARLES F. M. SAINT, C.B.E., M.D., M.B., F.R.C.S., F.R.A.C.S., Professor of Surgery, Cape Town University. Third edition. Demy 8vo. Pp. 367 + xii, with 231 illustrations. 1935. Bristol: John Wright & Sons Ltd. 15s. net.

PROFESSOR RUTHERFORD MORISON'S name is in itself a sufficient recommendation, but the appearance of the third edition is additional evidence of the usefulness of this book. The careful perusal of its pages, however, must arouse in the reader's mind certain criticisms and doubts with regard to the ideal method of presenting fundamental principles to the student beginning the study of surgery. When allowances have been made for the opinions of individual teachers, it must be admitted that any sound system should state the principles so clearly that the ideas conveyed to the student may be entirely free from ambiguity or confusion, that details should be introduced only to help to clarify these ideas, and that no error of terminology or fact should be permitted.

If the present volume is examined by this standard, it must be confessed that though an attempt has been made to simplify the work of the student by asking him to regard all pathological processes as "part of an all-embracing whole", the result is more closely akin to abstract philosophy than to the craft or even to the science of surgery. As the natural outcome of this system of unification, we are asked to regard an acute abscess as a spherical acute ulcer, and a sinus as a tubular ulcer, while syphilis, tuberculosis, and malignant disease

are grouped together in one chapter because of their close resemblance to one another. Though it is of fundamental importance to show that tissues respond to any kind of injury in a similar fashion, to infer that the variations in this response are so slight that all disease processes are identical is a hindrance rather than a help to the beginner.

Expressions of opinion are in many instances presented as statements of fact, there are several errors in terminology, more particularly in reference to malignant disease, and much of the detail is unnecessarily burdensome in a volume such as this. It may be that the authors have felt the necessity for including these minor points so that the book may compete with other short text-books on surgery for examination purposes, but it is regrettable that they should have been forced thereby to run the risk of obscuring the principles which it has been their chief object to enunciate.

The Modern Treatment of Burns and Scalds. By PHILIP H. MITCHINER, M.D., M.S. (Lond.), F.R.C.S., Hon. Surgeon to H.M. the King, etc. Crown 8vo. Pp. 64 + x, with 12 plates. 1935. London: Baillière, Tindall & Cox. 5s. net.

THIS admirable little book states with military precision how burns of all varieties should be treated. Its practical value is so great that it should be carefully studied by every medical student and practitioner, and its precepts should be drilled into all those responsible for first-aid treatment, especially in industrial establishments. As Mr. Mitchiner points out, there has been a tendency in the past to regard burns and scalds as of minor importance in surgery, but there is now no excuse for perpetuating the evil effects of this attitude towards injuries the mortality-rate of which can be reduced tenfold by proper methods of treatment.

Though the principal object of this book is to set forth these methods, a brief but adequate description is given of the pathological processes resulting from burns and scalds, and the superiority of a 2 per cent solution of tannic acid in water over all other local applications is explained on theoretical grounds as well as being proved by experience. Why 2 per cent is preferable to a stronger solution, the reason for the inclusion of perchloride of mercury in the formulæ for powders and tablets, and many similar practical questions, are fully considered, and the hints with regard to burns resulting from special agents, such as electricity, tar, cordite, petrol, phosgene, and mustard gas, are invaluable. Another evidence of the author's careful attention to detail is the table showing the doses of opium and morphia suitable for children and adults of all ages—a matter which is referred to rather vaguely in most text-books. Mr. Mitchiner has given much attention to the illustrations, which are very helpful, and for which the publishers also deserve a share of the praise.

Urology in General Practice. By ALEX. E. ROCHE, M.A., M.D., M.Ch. (Camb.), F.R.C.S., Assistant Genito-Urinary Surgeon, West London Hospital, etc. Demy 8vo. Pp. 356 + x, with 43 illustrations. 1935. London: H. K. Lewis & Co. Ltd. 17s. 6d. net.

THE late General Booth exclaimed, "Why should the devil have all the best tunes?" We might echo, "Why should writings on scientific subjects be so often dry as dust and repellent to even the eager reader"? That erudition is not of necessity allied with dullness has been shown by Huxley in the last century and more recently by Bland-Sutton, Moynihan, and Sir Arthur Keith.

Mr. Roche, perhaps influenced by these writers, has shown that the careful choice of words and the use of a well-restrained humour is not incompatible with a thorough knowledge of urology, and the result has been a most attractive and readable book. The one exception we take, though it may seem ungrateful after reading the book with such pleasure, is that the approach to the subject is that of the surgeon rather than that of the general practitioner; the latter might feel puzzled to find only eleven pages devoted to the study of the enlarged prostate, whilst no fewer than thirty-seven are filled with a discussion—a very able one, we admit—of torsion of the spermatic cord and of the hydatid of Morgagni. Whilst every general practitioner will see many cases of the former, he will be lucky if he sees more than one of the latter during the course of his career. The explanation is, of course, that

Mr. Roche has taken an enthusiastic interest in intrascrotal disease and has written much on this subject; moreover, he partially disarms the critic by his remark that the length of the chapters is not in proportion to the importance of the subjects discussed therein.

The chapters on "The Examination of a Urological Case", on "Hæmaturia and Dysuria", and on "Cystoscopy and Pyelography" are all admirable; so is that on "Urethral Catheters", though we feel surprised that a disciple of Sir John Thomson-Walker should advocate the use of tape for tying in a catheter; we thought that Thomson-Walker's rubber bridle had quite displaced this method as being clean and easier to adjust.

The author emphasizes his points by illustrative cases and by quotations; one of the most delightful of these is the *bon mot* of the physician who visited a ward of prostatic cases and remarked that he thought "many of them would stand a shave and hair-cut if it were done in two stages"; surely (to quote from Mr. Roche's favourite poet), "what oft was thought, but ne'er so well expressed", and a flattering unction to the souls of those surgeons who have encountered undeserved misfortune after prostatectomy.

We commend this book to those of our readers who wish to feel that they are keeping their knowledge of urology up to date, and like a volume that they can read with comfort in the train.

A Text-book of Fractures and Dislocations. Covering their Pathology, Diagnosis, and Treatment. By KELLOGG SPEED, S.B., M.D., F.A.C.S., Professor of Clinical Surgery, Rush Medical College of the University of Chicago. Third edition. Royal 8vo. Pp. 1000, with 1042 illustrations. 1935. London: Henry Kimpton. 50s. net.

THIS treatise on fractures has now earned a well-deserved reputation both in America and this country for its lucid and comprehensive description of this section of traumatic surgery. It opens with two chapters on the general principles of treatment and on operative treatment. These are perhaps the most valuable in the book, as they represent such a sound view of modern methods, accepting and describing all proved advances but not advocating any 'fads'. The detailed description of the first-aid outfit and the teaching by posters and slogans are very practical.

The bulk of the book is concerned with the description of individual fractures and dislocations and their treatment, with a great wealth of excellent illustrations, so that it forms an invaluable book of reference. In the matter of the use of unpadded plaster, of early active movements, and of the danger of applying massage in the early days, the author is not a follower of Böhler's teaching. For instance, spinal fractures are to be nursed for four to eight weeks in bed in hyperextension, and the importance of early active movements is not stressed. And in Colles's fracture the figure shows the hand in palmar flexion, and the splint is to be removed on the second or third day for movements and massage. In the same way Pott's fracture has the plaster case removed after three to four days and routine early walking is not advised. The treatise gives so many methods of treatment that the student lacks a clear guidance as to which to use in any given case.

Wilhelm Conrad Röntgen and the Early History of the Roentgen Rays. By OTTO GLASSER, Cleveland Clinic Foundation. Crown 4to. Pp. 494 + xii, with 94 illustrations. 1935. London: John Bale, Sons & Danielsson, Ltd. 32s. 6d. net.

THE appearance of this book ten years after Röntgen's death serves as a very timely reminder to the modern generation of the wonders of his discovery. Radiology has now taken such a dominating place in medical science that it is well to be able to look back and trace the development of this marvel.

Röntgen was born in Lennap in 1845, the son of a cloth merchant. His early student career was not particularly brilliant, and the facts that he was expelled from school (for refusing to inform about a misdemeanant) and that he failed in his 'Absolutorium' examination gave no promise of a great future. But when once he had devoted himself to pure physics, his industry and accuracy of observation soon gave him both reward and opportunity. Privat-docent at Strassburg, Professor of Physics at Giessen, then at Würzburg (1888), and finally at Munich (1901), were the posts which he filled.

It was on Nov. 8, 1895, that, working alone in his laboratory in Würzburg, he first observed the new rays from the illumination of some crystals of barium platino-cyanide by a Crookes tube which was covered with black paper. He published his discovery to the world on Dec. 18, 1895, in a preliminary communication, which he amplified early in the next year. There probably has never been a discovery which was more quickly acclaimed and appreciated all over the civilized world.

This book tells us the full story and also gives personal notes of this great scientist who combined in himself enthusiasm, industry, and modesty. There is also a complete account of the reception of the discovery by scientists and by the press both lay and medical. It concludes with a bibliography of 1044 references, a very large proportion of which were published in 1896.

The book is delightfully written in English and gives a good biographical sketch of the man and a lucid account of the genesis and growth of his discovery. We think, however, that its value would be increased if some chapters were added on the later development of radiology, whilst a good deal of the detailed repetitions of the early work might be omitted.

Röntgenology: the Borderland of the Normal and Early Pathological in the Skiagram. By ALBAN KÖHLER (Wiesbaden). Translated by ARTHUR TURNBULL, M.A., B.Sc., M.B., Ch.B. (Glasg.), Ex-Demonstrator of Anatomy, University of Glasgow. 7 in. × 10 in. Pp. 682 + xvi, with over 400 illustrations. 1935. London: Baillière, Tindall & Cox. 50s. net.

THE phrase 'German thoroughness' may perhaps be somewhat hackneyed, but one cannot avoid using this expression to describe one of the main characteristics of this unusually fine piece of work. The book is already familiar to English readers, the first English edition having appeared in 1928, thanks to the able efforts of Mr. Turnbull and others. It is a veritable mine of information, and its scope is well defined in the sub-title, "The Borderlands of the Normal and Early Pathological in the Skiagram."

In this new English edition the general arrangement has not been altered, but the subject matter has been suitably revised and expanded, and many references, together with frequent full summaries of the recent literature, are included. The author relies very largely upon clear line drawings to illustrate his points, although the number of actual skiagrams reproduced is considerable. In the course of the study of radiograms of the various parts of the body in large numbers of subjects, slight variations from what is generally considered the normal are constantly being encountered, and Professor Köhler's work can always be relied upon to solve such problems. In fact, to quote from Professor Fraser's remarks in his Preface, "it is difficult to conceive of any abnormality, or early pathological change demonstrable by X-ray, which is not considered in this excellent Treatise." It can be truly described as an authoritative and standard work, and, considering the enormous part played by radiography in medicine to-day, radiologists, physicians and surgeons, medico-legal experts, and students, should be eternally grateful to the author and the translator for making this volume available in the English language.

The Radiology of Bones and Joints. By JAMES F. BRAILSFORD, M.D. (B'ham.), M.R.C.S., Hunterian Professor, R.C.S., England, 1934–5; First President of the British Association of Radiologists, etc. Second Edition. Crown 4to. Pp. 57 + xxiii, with 340 illustrations. 1935. London: J. & A. Churchill Ltd. 30s. net.

THE production of the second edition following so quickly upon the issue of the first one is indicative of the very deserving success of this book. The present edition contains some additions in the text and illustrations, and the index has been reconstructed. There is a very complete discussion upon osteochondritis, voicing the many opinions which have been expressed concerning this subject, and the author has given his own conclusions based upon a very considerable experience. It is probable that the last word has yet to be written on this interesting subject. The section dealing with spondylolisthesis has been reconstructed and rendered

even more informative than it was in the previous edition. The chapter on bone dystrophies has been re-written and brought up to date. A brief description of dental radiology has been included, but it is very incomplete and does not compare at all favourably with the profundity of the rest of the book.

Dr. Brailsford has earned the gratitude of his fellow radiologists and deserves full praise for his very fine work.

The Theory and Practice of Anæsthesia. By M. D. NOSWORTHY, M.A., M.D., B.Ch. (Cantab.), Anæsthetist to Westminster Hospital, etc., with a Foreword by I. W. MAGILL, M.B., B.Ch. (Belfast), Anæsthetist to Westminster Hospital, etc. Crown 8vo. Pp. 223, with 35 illustrations. 1935. London: Hutchinson Scientific. 12s. 6d. net.

DR. NOSWORTHY'S large experience as an instructor in anæsthesia has enabled him to produce this book, which must prove a valuable guide to the novice, and, incidentally, contains much of real interest for the more experienced administrator.

While admitting that the administration of anæsthetics is largely an art, the author insists on the need for an insight into the scientific data upon which judgement may be based, and he describes at some length the recent views on the functions of oxygen and carbon dioxide in human physiology. Four chapters are devoted to theory, and the remaining twelve chapters to clinical material, with special attention paid to modern methods, the descriptions of technique being illustrated where necessary. Among the more recent developments the subject of premedication is described, with useful accounts of the various drugs in common use and the reason for selecting different drugs in particular cases. There obviously is not space in a book of this size to give details of the many ways in which regional anæsthesia may be employed, but under this heading is included a carefully written chapter on spinal anæsthesia, which should convince the reader of the care necessary if accidents are to be avoided and reliable results obtained.

The book cannot fail to become popular, and its readers will realize that the subject matter has been carefully digested and presented to them in an easily assimilable form. A feature of this little work is the thoroughness with which references have been quoted, also the clearness of the type and the neat style in which it has been produced.

A Guide to the Surgical Paper: With Questions and Answers. By R. J. MCNEILL LOVE, M.S. (Lond.), F.R.C.S., Surgeon, Royal Northern and Metropolitan Hospitals, etc. F'cap 8vo. Pp. 78. 1935. London: H. K. Lewis & Co. Ltd. 5s. net.

MR. MCNEILL LOVE'S small book will no doubt help many people. The plan on which it is founded is ingenious, and while it necessarily covers only a small proportion of the possible questions that may be set, it gives the candidate a scheme on which he may build up an answer to any parallel question. The published answers are necessarily condensed, and lists are made more use of than they should be in the ideal answer, but this book is to be commended, for one knows there is a type of candidate, by no means infrequent, to whom such a guide will be of the greatest possible assistance.

A Text-book of Surgical Pathology. By CHARLES F. ILLINGWORTH, M.D., F.R.C.S. (Ed.), Lecturer in Clinical Surgery, University of Edinburgh, Senior Assistant Surgeon, Edinburgh Western Hospital; and BRUCE M. DICK, M.B., F.R.C.S.(Ed.), Lecturer in Clinical Surgery, University of Edinburgh, Assistant Surgeon, Edinburgh Western Hospital. Second edition. Royal 8vo. Pp. 719 + x, with 301 illustrations. 1935. London: J. & A. Churchill Ltd. 36s. net.

THE authors set before themselves the task of writing a book on the pathology of surgical diseases on those aspects which are outside the scope of text-books on general pathology. In this object they have certainly succeeded, as is shown by the fact that the first edition of 1932 has been so soon followed by a second. There will be those who will doubt whether the necessary space should have been available for the introduction of paragraphs upon rare

conditions added to this edition, but there will be only one opinion upon the usefulness of re-writing certain sections. The authors certainly avoid the usual pitfall of clinicians when they undertake to write a volume of this nature on pathology—who mostly forget that their aim is pathology and not clinical surgery. The majority of the illustrations are well chosen and well produced, and the whole of the book is worthy of the house from which it emanates.

Diseases of the Liver, Gall Bladder, Ducts and Pancreas: their Diagnosis and Treatment. By SAMUEL WEISS, M.D., F.A.C.P., Clinical Professor of Gastroenterology, New York Polyclinic Medical School and Hospital, etc. With a chapter by J. P. GRANT, M.D., F.A.C.S., M.R.C.S., and A. J. QUIMBY, M.D., F.A.C.R. Imperial 8vo. Pp. 1099 + xxviii, with 358 illustrations and 6 coloured plates. 1935. New York: Paul B. Hoeber Inc. $10.00.

THE bulk of this large volume will be of more interest to the physician than the surgeon, and to the general practitioner rather than to either. It sets out to deal exhaustively with the practical side of biliary and pancreatic diseases. The chapter on surgery is limited to thirty-seven pages and deals with the preparation of the patient before operation, the standardized operations of cholecystostomy and cholecystectomy, and the removal of stones from the biliary ducts. At the end of the book is a bibliography running into close on 100 pages.

Surgical Nursing and After-treatment. A Handbook for Nurses and Others. By H. C. RUTHERFORD DARLING, M.D., M.S. (Lond.), F.R.C.S. (Eng.), F.R.F.P.S. (Glasgow), Surgeon, Prince Henry Hospital, New South Wales Hospital (Langton Clinic), etc. Crown 8vo. Pp. 738 + xii, with 187 illustrations. 1935. London: J. & A. Churchill Ltd. 9s. net.

THE higher examination standard now required for nurses is well met by this text-book, which at first sight, with its 738 pages, seems unnecessarily formidable. However, it reads clearly and simply, and not only gives the detailed technique of ordinary surgical nursing procedures but also seeks to elucidate them by giving an insight into surgical pathology and operative procedures and complications which may arise therefrom.

The illustrations are numerous and helpful, and the essentially practical nature of the whole book makes it suitable, not only as a guide to nurses in training, but also as a book of reference to those out of touch with hospital practice after their training is finished.

A Text-book of Surgery for Dental Students. By G. PERCIVAL MILLS, M.B., B.S. (Lond.), F.R.C.S., Hon. Surgeon, General Hospital, Birmingham, etc.; and HUMPHREY HUMPHREYS, O.B.E., M.C., T.D., K.H.P., M.B., Ch.B., M.D.S. (Birm.), L.D.S. (Eng.), Lecturer on Dental Anatomy, Birmingham University, etc. Fourth edition. Large 8vo. Pp. 342 + xii, with 63 illustrations. 1935. London: Edward Arnold & Co. 14s. net.

THIS book has proved its utility and popularity by the fact that it is now in its fourth edition. It gives a clear account of general surgical principles and of the special surgery of the mouth, jaws, and cranial and cervical regions. The illustrations, though not very numerous, are clear, and the book gives just what dental students require for their examination in surgery.

Estudos cirurgicos (First Series). By ENRICO BRANCO RIBEIRO (Santa Catharina). Large 8vo. Pp. 241. Illustrated. 1934. Sao Paulo, Brasil: Sociedade Editora Medica Limitada. No price given.

THE author dedicates his surgical studies to his teacher, Professor Montenegro. The subjects cover a wide field of general surgery, and show not only that the author is well read in the literature of the world, but that he has original views and is prepared to put them into execution. For example, in dealing with perforation of typhoid ulcers, he has found

it better to do a bold cæcostomy at the conclusion of his suturing operation rather than the ileostomy which is in favour in South America, and he describes his own technique for its performance. He has something to say about such different matters as the origin of gall-stones, the method of dealing with infections of the upper urinary tract, the proper management of cases of ovarian cyst, injuries to the skull involving the meninges, treatment of suppuration by vaccines, neurofibromatosis, cancer of the gall-bladder, and torsion of the spermatic cord. He gives an account of the construction and working of an emergency hospital provided by a power company for its very large list of employees. Altogether the book gives an interesting account of the work of an up-to-date surgeon in Brazil.

Diseases of the Thyroid Gland. By A. E. HERTZLER, M.D., Professor of Surgery, University of Kansas. Third edition. Royal 8vo. Pp. 348. Illustrated. 1935. London: Henry Kimpton. 32s. net.

THE author states that several new factors have intervened since the last edition was published. These chiefly concern the importance of goitre in connection with disease of the heart. He concludes that disease of the thyroid gland is often a continuous process, ending in cardiac death, and says that his experience leads him more and more to remove the whole gland in certain selected cases.

Anatomia della Lussazione congenita del' Anca. By V. PUTTI, Bologna. Pp. 334, with 77 plates, many in colour. Bologna: Licinio Cappelli. Lir. 300.

THIS is a very beautiful atlas, illustrating every phase of the anatomy of congenital dislocation of the hip. The figures, which are of great artistic merit, are drawings, photographs, and X-rays, and are made from dissections as well as from patients.

It would seem that the author, having by his work and experience become the greatest living authority on this subject, has set out to produce the most complete and perfect anatomical description and illustration. In this he has been eminently successful, and we can only express the wish that it may soon be translated into English and also that it may be followed by a companion volume dealing with the clinical aspect of this deformity.

Localized Rarefying Conditions of Bone. As exemplified by Legg-Perthes' Disease. Osgood-Schlatter's Disease, Kümmell's Disease and Related Conditions. By E. S. J, KING, M.D., D.Sc., M.S. (Melb.), F.R.C.S., F.R.A.C.S., Surgeon to Out-patients, Melbourne Hospital. Medium 8vo. Pp. 400 + xii, with 70 illustrations. 1935. London: Edward Arnold & Co. 35s. net.

THIS is a comprehensive monograph containing the most complete account yet published of the various types of osteochondritis juvenilis, post-traumatic rarefaction of bone, and the condition known as osteochondritis dissecans. It embodies the Jacksonian Prize Essay for 1933. As a work of reference it is invaluable. Our only criticism is that the author has perhaps tended to submerge his personal views on the etiology of the conditions, and that the introductory chapter on the structure, development, and physiology of bone is rather too episodic to be easily understood by the tyro. The illustrations, which come from the author's own collection, have been well chosen, and for the exhaustive list of references all surgeons will be grateful.

Bassini's Operation for the Radical Treatment of Inguinal Hernia. By Prof. ATTILIO CATTERINA, University of Genoa. Obl. Imperial 4to. Pp. 58, with 16 coloured plates by Orazio Gaigher, M.D. 1934. London: H. K. Lewis & Co. Ltd. 30s. net.

AN English edition of this atlas of plates illustrating Bassini's operation has been added to the already existing Italian, German, and French publications. For a full account of the work, readers are referred to the review of the German edition which appeared in this JOURNAL for January, 1934 (p. 555).

Collected Papers of St. Mark's Hospital, London: including a History of the Hospital. Centenary Volume, 1835–1935. Compiled by the Medical Committee. Crown 4to. Pp. 440 + xxvi, with 96 illustrations. 1935. London: H. K. Lewis & Co. Ltd. 30s. net.

IT was a happy thought that prompted the members of the Medical Committee of St. Mark's Hospital to commemorate the centenary of the hospital by publishing in book form a selection of the writings of past and present members of the staff. A glance at the voluminous contributions to the subject of diseases of the anus and rectum, which is to be found at the end of the book, shows how difficult it must have been to make the necessary selection. We congratulate the Medical Committee on having made an excellent choice, which is not only representative of the progress made in the treatment of rectal diseases throughout the century of the Hospital's existence, but is also indicative of the evolution of the present-day teaching at St. Mark's.

St. Bartholomew's Hospital Reports. Edited by LORD HORDER, W. G. BALL, R. G. CANTI, C. F. HARRIS, W. SHAW, H. H. WOOLLARD, R. C. ELMSLIE, G. EVANS, and J. P. ROSS. Vol. LXVIII. Demy 8vo. Pp. 351 + xxvi. Illustrated. 1935. London: John Murray. 21s. net.

FOLLOWING the practice which has been customary in their recent issues, this volume of the Saint Bartholomew's Reports for 1935 contains a series of papers on the urinary tract, particular attention being paid to infections. In these, Mr. Boyd's article on the anatomy and physiology of the kidney is most instructive. Mr. Girling Ball's contribution is a most comprehensive paper on the surgical aspect of the question, which is also dealt with in relation to pregnancy and in other aspects. Mr. Joseph Cunning contributes a sympathetic notice of the late F. A. Rose.

Reminiscences. By AGNES HUNT, D.B.E., R.R.C. Demy 8vo. Pp. 137. Illustrated. 1935. Shrewsbury: Wilding & Son Ltd. 5s. net.

THIS racy little book, which is introduced by a Foreword by the Hon. Sir Arthur Stanley, is both a delight and a disappointment. A delight because of the breezy and unconventional narrative of travel and adventure, voyages round the world, and a struggle with an early settler's difficulties in Australia. A disappointment because it gives such a meagre account of the great work which Dame Agnes Hunt began before the war, for the cure of various crippling conditions. Herself a life-long cripple, her wonderful pluck and enterprise will always prove that those who are physically handicapped can nevertheless do a great work for the betterment of the world.

Kleine Chirurgie. By Prof. Dr. HANS KURTZAHN. Third edition. Pp. 456, with 169 illustrations. Berlin and Vienna: Urban & Schwarzenberg. 1935. Paper covers, RM. 12; bound, RM. 13.50.

THIS is a book for the German student, dealing with minor and practical surgery. It is not likely to interest English students deeply.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Krebsproblem und praktische Chirurgie. By Prof. Dr. FRITZ KÖNIG (Wurzburg). Part 4 of *Vorträge aus der praktischen Chirurgie*, edited by Prof. Dr. ERICH LEXER (Munich). Royal 8vo. Pp. 27. 1935. Stuttgart: Ferdinand Enke. RM. 1.60.

Die Chirurgie der Hirngeschwülste. By Prof. Dr. N. GULEKE, Direktor der Chirurgischen, Univ.-Klinik, Jena. Part 5 of *Vorträge aus der praktischen Chirurgie*, edited by Prof. Dr. ERICH LEXER (Munich). Royal 8vo. Pp. 40. 1936. Stuttgart: Ferdinand Enke. RM. 2.20.

Der Schenkelhalsbruch: ein mechanisches Problem. By Dr. FRIEDRICH PAUWELS (Aachen). Royal 8vo. Pp. 157, with 186 illustrations. 1935. Stuttgart: Ferdinand Enke. Paper covers, RM. 13.60; bound, RM. 15.00.

Brain Preparations by Means of Defibrillation or Blunt Dissection. By Dr. J. WILH. HULKRANTZ, Professor of Anatomy, University of Uppsala, Sweden. Translated from the first German edition by HERBERT J. WILKINSON, Professor of Anatomy, University of Adelaide. Crown 4to. Pp. 48 + ix, with 48 illustrations. 1935. London: William Heinemann Medical Books Ltd. 10s. 6d. net.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London, etc. Fifth edition. Large 8vo. Pp. 287 + xii, with 341 illustrations. 1935. Bristol: John Wright & Sons Ltd. 21s. net.

The Early Diagnosis of Malignant Disease. By GEOFFREY KEYNES, M.A., M.D. (Cantab.), F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital, etc. F'cap 8vo. Pp. 70. 1935. London: John Bale, Sons & Danielsson, Ltd. 2s. 6d. net.

Tumours of the Urinary Bladder. By EDWIN BEER, M.D., F.A.C.S., Visiting Surgeon, Mt. Sinai Hospital; Consulting Surgeon, Bellevue Hospital, New York City. Large 8vo. Pp. 166 + vi, with 52 illustrations (8 in colour). 1925. London: Baillière, Tindall & Cox. 16s. net.

The Early Diagnosis of the Acute Abdomen. By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S., Surgeon to St. Mary's Hospital, Paddington, etc. Seventh edition. Medium 8vo. Pp. 254 + xiv, with 33 illustrations. 1935. London: Humphrey Milford, Oxford University Press. 10s. 6d. net.

Chirurgische Operationslehre. By Dr. MAX SAEGESSAR, Privatdozent an der Universität Bern. Crown 8vo. Pp. 189 + vi, with 132 illustrations. 1935. Berlin: Julius Springer. Paper covers, RM. 7.50; bound, 8.60.

The Principles and Practice of X-ray Therapy. By FFRANGCON ROBERTS, M.A., M.D. (Cantab.), M.R.C.P. (Lond.), D.M.R.E. (Cantab.), Physician in Charge of the Douty X-ray Clinic, Addenbrooke's Hospital, Cambridge. Demy 8vo. Pp. 214 + xii, with 110 illustrations, including 6 plates. 1936. London: H. K. Lewis & Co. Ltd. 10s. 6d. net.

The Foot. By NORMAN C. LAKE, M.D., M.S. (Lond.), F.R.C.S., Senior Surgeon and Lecturer on Surgery, Charing Cross Hospital. Large 8vo. Pp. 330 + vii, with 95 illustrations. 1935. London: Baillière, Tindall & Cox. 12s. 6d. net.

Post-graduate Surgery. Edited by RODNEY MAINGOT, F.R.C.S., Senior Surgeon to the Royal Waterloo Hospital, etc. With an Introduction by the Right Hon. LORD MOYNIHAN OF LEEDS, K.C.M.G., C.B., M.S., F.R.C.S. In three volumes. Royal 8vo. Vol. I: Pp. 1742 + xvi, with 846 illustrations. 1936. London: Medical Publications Ltd. £6 6s. the set, or £2 10s. per volume.